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THE PROBLEM OF GASTRIC CARCINOMA.[1]

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THOUGH commonplace to all surgeons of experience, it is apparently unknown to the general public that cancer in its early and later stages behaves towards treatment as if it were two entirely different diseases. Notwithstanding the fact that the Department of Public Health of New South Wales has broadcast posters bringing the subject of cancer, especially its early symptoms and signs, before the public, cancer of the stomach, with rare exceptions, is seen by the surgeon only in its more advanced stages, when the diagnosis is obvious and the results of treatment problematical. For instance, among the twenty-two cases of this malady referred during the past twelve months, only in six was it possible to carry out the modern operation of partial gastrectomy, and none of these could be termed "early cases".

This state of affairs, however, is a distinct improvement on that pertaining to the period prior to the establishment of an X ray department at the State Hospital, when, with one exception, no case was seen sufficiently early to permit the undertaking of partial gastrectomy.

There are two phases to the period which ends with the commencement of surgical treatment. The first lasts from the appearance of the first sign of cancer to the first medical examination, usually made by a general practitioner. The second phase dates from this examination until expert surgical treatment is instituted.

[1] Read at a clinical meeting of the staff of the New South Wales Community Hospital on February 23, 1933.

Probably there is no surgical malady for which the general practitioner can accomplish more than carcinoma of the stomach; for he must necessarily be the one to see these cases in their early stages.

On the other hand, it is interesting to note that when forty-one consecutive case histories of cancer of the stomach occurring amongst medical practitioners were analysed by W. C. Alvarez, symptoms had already been present prior to examination, on the average, for at least eleven months.

A potent factor in bringing about this unsatisfactory state of affairs is the fact that the cardinal symptoms of this malady, as described in the current standard text books, are those of the well established disease, with organic changes so firmly rooted that there is little chance of complete removal of the growth.

Although wasting, cachexia, anæmia, vomiting and tumour form a syndrome so plain that he who runs may read, when the histories of such cases are reviewed, there are generally to be brought to light symptoms which have existed, treated or untreated, for a considerable number of months, and which should have aroused suspicion. It therefore follows that if the diagnosis of carcinoma of the stomach is to be made sufficiently early to permit the effective performance of the modern operation of partial gastrectomy, these early clues should be studied and the suspicion of carcinoma verified by X ray and chemical tests. Furthermore, the public should be taught to realize that it is far more important to ascertain the cause of a chronic dyspepsia in a middle-aged person than to relieve it. Only by the adoption of such methods can effective war be waged against this malady.

The Incidence of Carcinoma Ventriculi.

Of all fatal cases of cancer, *carcinoma ventriculi* has been found to be easily the commonest, constituting a percentage of total cancer deaths varying in New South Wales from 25% to 26%, as compared with 35% to 40% in European countries. It causes annually more than two and a half times as many deaths as cancer of the breast, and three times as many as cancer of the uterus. Each year between 1,500 and 1,600 deaths are recorded in the Commonwealth of Australia as being due to this disease.

In the State of New South Wales the death rate from cancer of the stomach is approximately 24 to 25 per 100,000 of population. In Table I are recorded the mortality figures for the years 1928 to 1932.

TABLE I.

Showing Mortality from Carcinoma Ventriculi in New South Wales. (Stomach, Duodenum and Pylorus.)

Year.	Males.	Females.	Total Number of Persons.	Rates per 100,000 of Population.	Total Cancer Deaths.	Percentage of Deaths from Cancer of Stomach to Deaths from all Forms of Cancer.
1928	388	215	603	24·9	2,280	26·4
1929	392	189	581	23·6	2,322	25·0
1930	386	217	603	24·2	2,290	26·3
1931	418	207	625	24·9	2,439	25·6
1932	438	190	628	24·8	2,534	24·8

The sex incidence of carcinoma of the stomach is clearly demonstrated in Table I.

Early Symptoms.

At the outset the supreme importance of obtaining a detailed and accurate history of every case should be emphasized. Cancer of the stomach, in the majority of cases, produces symptoms early; but they are usually vague and easily overlooked; those supposed to be typical of carcinoma only reveal themselves at a late stage of the disease.

Edmund Spriggs carefully analysed the symptoms in thirty-eight consecutive proven cases of cancer of the stomach, and found that the average order of frequency in which the symptoms first appeared was as follows: (i) discomfort or pain in the upper part of the abdomen, not related to food; (ii) loss of appetite or dislike of food, and nausea; (iii) pain or discomfort after food; (iv) heartburn

TABLE II.

Showing Anatomical Sites of Cancer and Mortality, from 1928 to 1931, in New South Wales.

Causes of Death.	1928.			1929.			1930.			1931.		
	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.
Cancer of the:												
Buccal Cavity and Pharynx ..	142	11	153	131	16	147	131	13	144	128	11	139
Digestive Organs and Peritoneum—												
A. Œsophagus	48	10	58	55	14	69	61	14	75	47	15	62
B. Stomach and Duodenum ..	388	215	603	392	189	581	386	217	603	418	207	625
C. Rectum	51	45	96	58	38	96	49	49	98	53	43	96
D. Liver and Biliary Ducts.. ..	75	90	165	64	71	135	60	66	126	52	76	128
E. Pancreas	21	13	34	36	17	53	40	15	55	38	21	59
F. Peritoneum	3	8	11	9	9	18	4	6	10	2	7	9
G. Intestines	138	157	295	135	152	287	134	158	292	134	159	293
H. Other Organs	—	—	—	—	—	—	—	1	1	—	—	—
Respiratory Organs	61	14	75	49	25	74	59	18	77	82	20	102
Uterus	—	164	164	—	170	170	—	162	162	—	203	203
Other Female Genital Organs	—	38	38	—	65	65	—	57	57	—	65	65
Breast	1	191	192	3	190	193	4	193	197	2	231	233
Male Genito-urinary Organs	136	—	136	141	—	141	159	—	159	177	—	177
Skin	50	31	81	52	32	84	48	30	78	43	21	664
Other or Unspecified Organs	82	97	179	99	110	209	77	79	156	90	94	184
Total Cancers	1,196	1,084	2,280	1,224	1,098	2,322	1,212	1,078	2,290	1,266	1,173	2,439

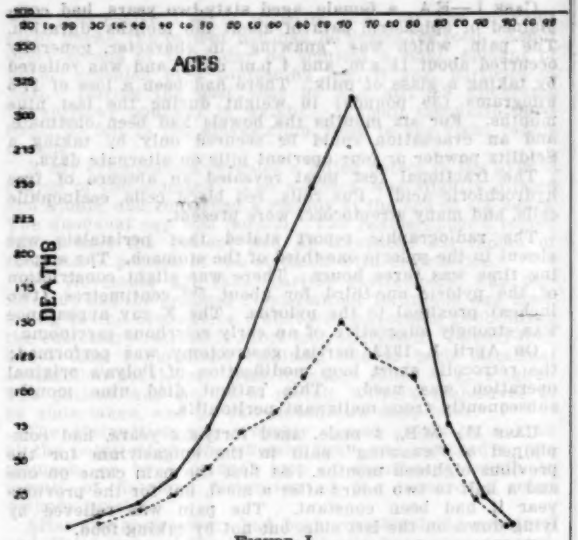

FIGURE I.

Graph showing age groups and mortality from cancer of the stomach, from 1928 to 1931, in New South Wales. Total cases, 2,384 (males 1,561, females 823). Continuous line denotes males, interrupted line, females.

and eructation; (v) flatulence; (vi) vomiting; (vii) regurgitation of mucus; (viii) weakness and constipation; (ix) loss of weight; (x) dysphagia or inability to take solids; (xi) tumour.

TABLE III.

Showing Position of Origin in One Hundred and Twenty Cases of Carcinoma of the Stomach (Stewart).

Pylorus and prepyloric inch	67·0%
Cardiac end	16·5%
Lesser curvature region	11·5%
Rest of stomach	5·0%

Usually the first symptom noted by the patient is some disturbance in the upper part of the abdomen; he is conscious of "having a stomach", whereas formerly he was unaware of the existence of this organ, could eat anything, and never gave his digestion a thought. It is comparatively rare to get any previous history of digestive trouble, although occasionally this is found. A sense of fullness and discomfort occurs after meals, and tends to persist throughout the day, and the patient thinks his food is not agreeing with him. Soon the discomfort and uneasiness are accompanied by flatulence and belching of gas, at first following ingestion of food, but, as the severity increases, becoming more or less constant. At a later period the patient may complain of pain, described as of a dull, sickening character, referred to the epigastrium, but rarely of the severity experienced in gastric ulcer. Loss of appetite is gradual and insidious in its onset. The patient refuses food, not because it brings on pain, but because his appetite has gone. In many cases there is a marked aversion to meat and fat. Vomiting is rarely marked, and frequently amounts only to regurgitation of a small amount of fluid, which the patient describes as being foul or offensive in character. Progressive loss of strength is an early symptom, and is often associated with impaired mental activity and depression of spirits.

As regards the duration of symptoms, Berger, of Vienna, points out that there are three types of history.

In the first type (66%) there is a short history of gastric symptoms of from four to five months' duration; in the second, a long history, between six and twenty-four months (18%), and over two years (14%). In the third type (2%) there are no gastric symptoms at all.

Types of Carcinoma Ventriculi.

It is obvious from the above brief analysis that no single description can include all cases of gastric carcinoma, because the symptoms are so multiform and the duration so uncertain. Lord Moynihan has endeavoured to group these cases into four distinct types: the duodenal ulcer type, the dyspeptic type, the obstructive type, and the hæmorrhagic type.

The Duodenal Ulcer Type.

In some cases the symptoms closely resemble those of duodenal ulcer. The patient has hunger pain, but there are no remissions, and symptoms are not relieved by the customary medical treatment. This group may be enlarged to include those cases in which the symptoms of a gastric ulcer slowly and gradually change to those of a carcinoma. In other words, the pain becomes more continuous, though less severe, there is loss of periodicity throughout the day, the appetite begins to fail, and the vomiting may alter in character and does not relieve the pain.

Professor M. J. Stewart's conclusions, that 9·5% of cases of chronic ulcer of the stomach become malignant, and 17% of cancers originate in chronic ulcer, are probably somewhere near the truth. Orator's investigations at the Eiselburg Clinic on this subject are interesting, in that he found that, while one-third of prepyloric ulcers underwent cancerous degeneration, only 3% of those situated in the *pars media* did so. Almost a decade ago Stewart pointed out that, whereas 73% of chronic ulcers and 11·5% of cancers originated at the lesser curvature, the pylorus and prepyloric inch were responsible for the origin of not only 17·5% of chronic ulcers, but also 67% of all cases of gastric carcinoma. Further, in ten cases in which cancer had apparently originated in a preexisting ulcer, nine were on the lesser curvature and only one near the pylorus.

The historical example of a gastric ulcer undergoing malignant degeneration is the illness of Napoleon Bonaparte. Gastric pain first became prominent about 1807, when the Emperor was thirty-eight years of age. In August, 1813, at Dresden, when he might have followed up the Allies and routed them, he was seized with a violent attack of "gastric spasm". By 1820 the illness had gained ground. "His stomach burned like fire, and in some of the paroxysms he rolled on the floor in agony. He would shiver with cold, and hot fomentations could

never be hot enough for him." Malignant degeneration supervened somewhere about September, 1820. Three weeks before his death, wasting and cachexia became more apparent and pain almost continuous. On April 29, 1821, the Emperor had a severe rigor and sank into delirium, which, except for a short lucid interval, persisted until his death at sunset (5.49 p.m.) on May 5, 1821. In his will appears the following passage: "I expressly demand that a post-mortem shall be made and in especial that the stomach shall be examined. I believe that I am dying of the same illness as my father." Napoleon's grandfather, father, uncle, brother Lucien, and sister Caroline all died of cancer.

The following is the wording of a manuscript copy, in possession of Professor Sherran, Professor of Surgery in the University of Aberdeen, originally written in 1821, at Longwood, in the Island of Saint Helena, by one of the doctors actually present at the *post mortem* examination on the Emperor Napoleon.

Longwood,
Saint Helena,
May 6th, 1821.

REPORT OF APPEARANCES ON DESECTION OF THE BODY OF NAPOLEON BONAPARTE.

On a superficial view the body appeared very fat, which state was confirmed by the first incision down its centre, where the fat was upwards of one inch thick over the sternum, and one inch and a half over the abdomen.

On cutting through the cartilages of the ribs and exposing the cavity of the thorax a trifling adhesion of the left pleura was found to the pleura costalis.

About three ounces of reddish fluid were contained in the left cavity and nearly eight ounces in the right.

The lungs were quite sound.

The pericardium was natural and contained about an ounce of fluid.

The heart was of the natural size but thickly covered with fat.

The auricles and ventricles exhibited nothing extraordinary except that the muscular parts appeared rather paler than natural.

Upon opening the abdomen the omentum was found remarkably fat, and on exposing the stomach that viscus was found the seat of extensive disease, strong adhesions connected the whole superior surface, particularly about the pyloric extremity to the concave surface of the left lobe of the liver, and on seperating these an ulcer which penetrated the coats of the stomach was discovered one inch from the pylorus sufficient to allow the passage of the little finger.

The internal surface of the stomach to nearly its whole extent, was a mass of cancerous disease or schirrous portions advancing to cancer, this was particularly noticed near the pylorus. The cardiac extremety for a small space near the termination of the asophagus was the only part appearing in a healthy state. The stomach was found nearly filled with a large quantity of fluid resembling coffee.

The convex surface of the left lobe of the liver adhered to the diaphragm. With the exception of the adhesions occasioned by the disease in the stomach no unhealthy appearance presented itself in the liver.

The remainder of the abdominal viscera were in a healthy state.

A slight peculiarity in the formation of the left kidney was observed.

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CASE I.—E.A., a female, aged sixty-two years, had complained of epigastric pain of about ten months' duration. The pain, which was "gnawing" in character, generally occurred about 11 a.m. and 4 p.m. daily, and was relieved by taking a glass of milk. There had been a loss of 17·5 kilograms (39 pounds) in weight during the last nine months. For six months the bowels had been obstinate, and an evacuation could be secured only by taking a Seidlitz powder or four aperient pills on alternate days.

The fractional test meal revealed an absence of free hydrochloric acid. Pus cells, red blood cells, eosinophile cells, and many streptococci were present.

The radiographic report stated that peristalsis was absent in the pyloric one-third of the stomach. The emptying time was three hours. There was slight constriction of the pyloric one-third for about 5·0 centimetres (two inches) proximal to the pylorus. The X ray appearance was strongly suggestive of an early scirrhous carcinoma.

On April 1, 1932, partial gastrectomy was performed; the retrocolic short loop modification of Polya's original operation was used. This patient died nine months subsequently from malignant peritonitis.

CASE II.—M.B., a male, aged forty-six years, had complained of "gnawing" pain in the epigastrium for the previous eighteen months. At first the pain came on one and a half to two hours after a meal, but for the previous year it had been constant. The pain was relieved by lying down on the left side, but not by taking food.

For the past six months the pain had frequently awakened the patient, usually about one or two o'clock in the morning. During this period there had been progressive loss of appetite, and now the patient had little or no desire for food. During the previous year he had lost 6·3 kilograms (fourteen pounds) in weight.

A tender, freely movable tumour, the size of a pigeon's egg, could be felt in the epigastrium deep to the right rectus muscle. X ray examination showed a constant filling defect on the greater curvature, which on palpation revealed a persistent fleck of barium. The duodenal cap was normal, and there was no residue at the end of four and a half hours. The radiographer's report stated that the patient had some type of ulceration of the stomach, probably simple rather than malignant.

Partial gastrectomy was performed; the customary short loop modification of the original Polya operation was used. Examination of sections of the mass showed the tumour to be a columnar-celled carcinoma. Sixteen months later this patient was alive and well, and acting as "rouseabout" to Ward XVI.

The Dyspeptic Type.

As 80% of all cases of gastric cancer occur in patients between the ages of fifty and eighty, persistent and progressive indigestion at this period of life should always be regarded with suspicion, especially if it be of sudden onset, for, as Hale White pointed out, "if symptoms of serious chronic gastric indigestion first appear after the age of forty, organic disease of the stomach should be strongly suspected". Hence, if adequate medical treatment fails to cure this dyspepsia within fourteen days, an immediate investigation by means of a test meal and radiography should always be carried out. Horder goes further and teaches that laparotomy should be performed in every gastric case, irrespective of the patient's age, whenever there is a failure to gain weight or to maintain weight after the end of the third week of efficient treatment.

CASE III.—P.C., a man, aged eighty years, had complained of epigastric pain of ten weeks' duration. The pain was nagging in character, occurred about ten minutes after a meal, and persisted for approximately half an hour. It was not relieved by vomiting. Associated with the pain

was progressive loss of appetite, and, for the previous eight weeks, the patient had had no desire for food. About every second or third day he vomited a small amount of bitter, greenish fluid containing few, if any, particles of food. During the past year he had lost 9·9 kilograms (twenty-two pounds) in weight. A small indefinite mass could be felt in the epigastrium, deep to the right rectus muscle.

X ray examination revealed a filling defect in the pyloric region of the stomach. The stomach was slightly dilated and atonic, and contained a residue at the end of six hours. The duodenal cap was normal. The radiographer's report stated that the patient had an advanced carcinoma of the pyloric portion of the stomach with obstruction.

On November 23, 1932, partial gastrectomy was performed; but the original Polya-Balfour technique was used, owing to the diminutive transverse mesocolon prohibiting the customary retrocolic short loop operation. Twenty-four hours later regurgitant vomiting due to high intestinal obstruction developed. Gastric lavage failed to give relief. The patient was too ill to permit Witzel's manœuvre to be undertaken, and he succumbed two days later.

The obstruction was probably caused either by a kink at the point where the efferent limb was attached to the stomach, or by a jejunal loop of insufficient length to permit of flatulent distension of the transverse colon without tension.

After the gastrectomy had been completed the proximal and distal loops of the jejunum should have been approximated at a point on a level with the duodeno-jejunal flexure and entero-anastomosis performed.

CASE IV.—D.C., a man, aged fifty-one years, had complained of vague epigastric symptoms, unrelated to food, for the past ten months. Occult blood had been present in the stools, and a provisional diagnosis of duodenal ulcer had been made.

By the use of a modified Sippy's diet he had gained 1·3 kilograms (three pounds) in weight in a month; but his indigestion had not left him.

For the past seven months he had had discomfort and fullness after meals, for which he frequently sought relief by inducing vomiting. During the past three months his appetite had entirely disappeared. Examination of the stomach contents after a fractional test meal revealed the absence of free hydrochloric acid and the presence of lactic acid. Blood was present in the specimens removed at one and one and a half hours, as well as in the fasting residue.

X ray examination revealed a filling defect of the pyloric antrum. The stomach was not dilated and there was no residue at the end of four and a half hours. The duodenal cap was normal. The radiographer stated that the patient had a pyloric neoplasm.

Partial gastrectomy was performed by the short loop modification of Polya's original operation. This patient left hospital and has since been lost sight of.

The Obstructive Type.

The symptoms produced by the obstructive type are those of obstruction at either end or in the body of the stomach. When the disease commences at the pylorus it usually causes obstruction, and the symptoms then are no longer characteristic of a carcinoma, but are those of mechanical obstruction; that is to say, discomfort and fullness coming on immediately after meals, loss of appetite due to secondary gastritis, and the vomiting of large amounts of fermented fluid at relatively long intervals.

CASE V.—D.R., a fireman, aged sixty-two years, stated that he had been having attacks of vomiting for the previous nine months. At first the vomiting had been infrequent, but for the past six months it had occurred at least once daily, more frequently in the evening after tea. No actual pain was experienced, but a feeling of discomfort and fullness, which came on almost immediately after meals. There had been marked loss of appetite during the past six months, and now he had little or no desire for food. During his illness he had lost 3·1 kilograms (seven pounds) in weight.

X ray examination revealed a filling defect in the region of the pyloric antrum. The stomach was not dilated, but there was a moderate residue at the end of four and a half hours. The duodenal cap was normal. The radiographer considered the patient had a pyloric neoplasm.

Partial gastrectomy was performed on December 9, 1932, by the retrocolic short loop modification of Polya's original operation. Convalescence was uneventful until the evening of the seventh day, when the patient suddenly collapsed and died one hour later. The provisional diagnosis was pulmonary embolism.

CASE VI.—J.B., a man, aged sixty-one years, was seen in July, 1932. For the previous two years he had been complaining of epigastric pain at irregular intervals after food. The appetite was good, but food gave rise to epigastric distension and belching. Vomiting of a large quantity of fluid occurred about twice a week, but did not relieve the pain. He had lost 12·6 kilograms (twenty-eight pounds) in weight in the past twelve months. Hæmatemesis and melæna had never been noticed.

X ray examination revealed a filling defect of the pylorus. The stomach was not dilated and there was no residue at the end of five and a half hours. The duodenal cap was normal. The radiographer's report was to the effect that the patient had a neoplasm (malignant) of the pylorus, but that no obstruction was present.

Partial gastrectomy was performed by the retrocolic short loop modification of Polya's original operation. This patient was a working or yard patient until October 26, 1932, when he was transferred back to Liverpool State Hospital.

The Hæmorrhagic Type.

The hæmorrhagic type, which is marked by severe or slight hæmatemesis, is rare.

One is struck by the duration of time that is allowed to elapse between the onset of suspicious symptoms and adequate radiological and chemical examination of the viscus. Thaysen mentions that, in hospital, patients have usually been treated for six or more months. In my own cases the intervals in those cases in which partial gastrectomy was performed were ten months, eighteen months, ten weeks, ten months, nine months, and two years, respectively; in the other cases of this series the average time amounted to eighteen and a half months.

Tests.

The early symptoms suspicious of carcinoma having been enumerated, the means at our disposal of proving or disproving their correctness must be investigated.

Chemical Tests.

The Test for Occult Blood.—Occult blood, as detected by Gregersen's modification of the benzidine test, is present in 75% of cases of carcinoma of the stomach (Carson).

This test is extremely simple, and may be carried out by anybody possessing an elementary knowledge of chemistry. Repeated examinations are desirable, and the more frequently the test is made, the more likely are the numbers of positive results to increase. As a confirmatory test the stools may be examined spectroscopically for traces of hæmatoporphyrin. Occult blood due to chronic gastric ulcer usually disappears from the stools during treat-

ment; but this does not usually occur when it is caused by carcinoma.

Gastric Analysis.—For purposes of gastric analysis a test meal is given. The patient is given a light supper, consisting of a cup of milk containing two teaspoonfuls of charcoal or two charcoal biscuits, and the examination is undertaken on the following morning before any further food or drink has been taken.

The test meal is made by boiling two tablespoonfuls of oatmeal in a quart of water until the total volume is reduced to one pint; the mixture is then strained through muslin or a fine wire strainer. Salt must not be used as flavouring, as often the pathologist wishes to analyse the chloride content of the stomach contents.

In the investigation of carcinoma of the stomach by means of test meals special stress should be placed upon: (*a*) achlorhydria, (*b*) the presence of lactic acid, (*c*) the presence of blood, and (*d*) evidence of stagnation.

In the past, considerable stress has been laid upon absence of free hydrochloric acid and diminution of total acidity in the gastric contents. Such a reading is usually obtainable when carcinomata have originated in a previously healthy stomach; but there are so many exceptions that too great importance must not be attached to it. In cases of carcinomata in which the gastric symptoms have extended over many years, the acidity, both free and total, may approximate that customary with chronic ulcer; but when an extended series of test meals has been undertaken throughout the illness, a marked fall in the readings will be noted, even though the figures may still be above the normal.

Achlorhydria, especially if associated with the presence of lactic acid, is strong presumptive evidence of carcinoma; in fact, such a dual finding is said to occur in no other surgical condition, with the exception of cancer of the œsophagus (Dunlop). It has, however, been questioned whether such a finding may be regarded as an aid towards early recognition. The constant presence of blood in association with achlorhydria points to the lesion being a carcinoma. The achlorhydria of chronic gastritis or chronic gastric ulcer is alleged to differ from that of carcinoma in that, if the test meal be repeated after preliminary gastric lavage, the achlorhydria is replaced by a hypochlorhydria.

As approximately 65% of gastric carcinomata originate in the pylorus and prepyloric segment, it follows that one of the early signs of this malady may be impairment of gastric motility. The presence of any of the previous night's supper in the fasting contents of the stomach points to gastric stagnation.

Radiological Examination.

The main inquiry really hinges upon the X ray examination; for it must necessarily follow that the radiologist's approach to accuracy must be far greater than that of either the clinician or the pathologist.

Useful signs in the early stages are loss of flexibility and absence of peristaltic waves over a small area of the stomach wall, more often in the *pars pylorica*. Peristalsis ceases at the point where the muscular coat is replaced by neoplastic tissue.

Carcinomatous ulcers may cause difficulty, but when seen in profile the crater rarely projects beyond the plane of the gastric mucosa, so that a true niche is not present; furthermore, the edges are raised above the surrounding mucous membrane. If the carcinoma be small and has not yet infiltrated the muscular coats of the stomach, then neither defect in contour nor abnormality in peristalsis will of necessity be evident.

There are, however, two schools of radiology: one which favours the full opaque meal, and the other the striæ meal. Haudek has drawn attention to the fact that, by ingesting a small quantity of barium suspension, the folds of the gastric mucosa may be thrown into relief. "Central filling defects" (mucosal swellings) and interruptions of the mucosal "relief pattern" then become useful signs in the early diagnosis of carcinoma. Although certain Continental observers claim that there are special types of mucosal relief pattern indicating a predisposition to gastric ulcer and carcinoma respectively, this observation is at present barren as regards clinical value.

Other Tests.

Gastroscopy.—Gastroscopy, as Bland-Sutton puts it, requires the instinct of a sword swallower and the eye of a hawk. The gastrophor has proved unreliable, and only limited areas of the stomach can be photographed.

The Wassermann Test.—In every suspected case of cancer of the stomach a routine Wassermann test and a full blood count should be carried out. As every inmate of the State Hospital is subjected to a routine Wassermann test, and as there have been 577 positive results out of 6,456 cases, approximately 8·8%, I consider myself fortunate that the pathologist has not yet returned one of my operation specimens labelled "gastric syphilis".

Operation.

Cancer of the stomach can be cured only by operation.

Whilst many technical variations are possible, certain general principles, determined by the mode of spread of the disease, must be held inviolable. The aim of the operation is not merely to remove three-quarters of the viscus, but to remove intact the permeated area of lymph-vascular tissue which surrounds the primary growth, in one piece with the lymphatic glands, which may have been embolically invaded along the trunk lymphatics of the permeated area.

Except in Schoemaker's operation, the stomach is divided across in a straight line, and the continuity of the alimentary canal is restored by termino-lateral gastro-jejunostomy.

In the original Polya operation a long loop of jejunum was brought up through an opening in the

transverse mesocolon, but in Balfour's modification the loop was diverted anterior to the transverse colon.

The Polya-Balfour gastrectomy has undergone a further modification whereby, instead of the antecolic long loop, thirty to thirty-five centimetres (twelve to fourteen inches) in length, a short loop of jejunum, only three or four inches in length, is brought up to the stomach to the left of the splenic flexure, thus enabling the stoma to lie transversely under the left cupola of the diaphragm.

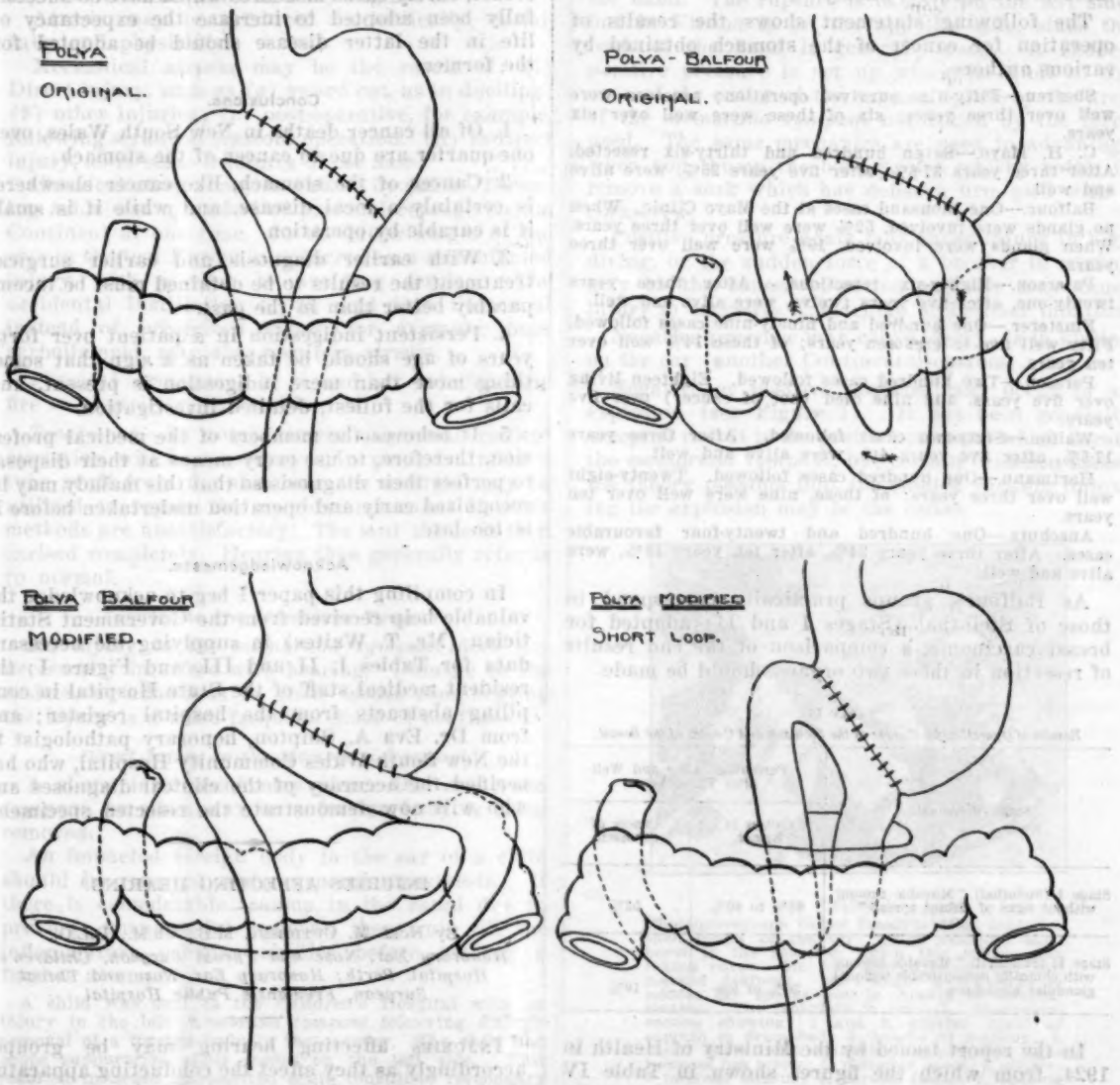

FIGURE II.

With one exception, in which the transverse mesocolon was so short and contracted as to prohibit a retrocolic anastomosis, the original Polya operation, modified by the use of a short loop to enable the anastomosis to be made four or five inches from the duodeno-jejunal flexure, has been adopted in this series.

In the original Polya operation the oral end of the loop was attached to the lesser curvature, so that the duodenal contents might enter the upper margin of the stomach and thus protect the jejunal mucosa from the action of the gastric juice. Gastric chemistry has apparently altered since those days; further, owing to the short portion of stomach remaining, one frequently has had to reverse this orientation and no ill effect has eventuated.

The results of this operation are encouraging. The primary mortality should not exceed 18%.

At the present time about 70% of those who have

survived the operation die of recurrences within three years. We must, however, remember that these are late cases. The average duration of life, if recurrence takes place, is eighteen months. The quality of life is good; as a rule there is no indigestion or vomiting, and the recurrence usually does not affect the stomach. In other words, the patients are given one or two years of happy life in place of three to six months' misery.

The following statement shows the results of operation for cancer of the stomach obtained by various authors.

Sherren.—Fifty-nine survived operation; nineteen were well over three years; six of these were well over six years.

C. H. Mayo.—Seven hundred and thirty-six resected. After three years 37·6%, after five years 25%, were alive and well.

Balfour.—One thousand cases at the Mayo Clinic. When no glands were involved, 52% were well over three years. When glands were involved, 19% were well over three years.

Paterson.—Eighty-six resections. After three years twenty-one, after five years twelve, were alive and well.

Finsterer.—One hundred and ninety-nine cases followed. Fifty well five to eighteen years; of these 14% well over ten years.

Persson.—Two hundred cases followed. Eighteen living over five years, and nine died (not of cancer) over five years.

Walton.—Sixty-two cases followed. After three years 17·5%, after five years 4%, were alive and well.

Hartmann.—One hundred cases followed. Twenty-eight well over three years; of these, nine were well over ten years.

Anschutz.—One hundred and twenty-four favourable cases. After three years 34%, after ten years 13%, were alive and well.

As Balfour's groups practically correspond to those of Steinthal (Stages I and II) adopted for breast carcinoma, a comparison of the end results of resection in these two organs should be made.

TABLE IV.

Results of Operation for Cancer of the Stomach and Cancer of the Breast.

Stage of Growth.	Percentage Alive and Well after Three Years.	
	Cancer of Breast.	Cancer of Stomach.
Stage I (Steinthal) "Movable tumour without signs of distant spread" ..	65% to 80%	52%
Stage II (Steinthal) "Movable tumour with clinically demonstrable regional glandular metastases"	30% or less	19%

In the report issued by the Ministry of Health in 1924, from which the figures shown in Table IV were taken, Dr. Lane-Claypon goes on to state: (i) That the expectation of life after the onset of cancer of the breast, if the disease is allowed to run its own course, may be taken as being on an average not more than three years and six months. (ii) That in the aggregate, that is, without reference to the stage of the disease at operation, the effect of the "complete" or modern operation is to prolong life, increasing the expectation of life from the onset of the disease by two or three years. (iii) That of patients operated upon by the "complete" operation while the disease is still local, the expectation of life from the onset of the disease may be on the average as much as ten years more than that of persons not treated by operation.

Since the death rate from cancer of the stomach is two and a half times that of carcinoma of the breast, surely those measures which have so successfully been adopted to increase the expectancy of life in the latter disease should be adopted for the former.

Conclusions.

1. Of all cancer deaths in New South Wales, over one-quarter are due to cancer of the stomach.

2. Cancer of the stomach, like cancer elsewhere, is certainly a local disease, and while it is small it is curable by operation.

3. With earlier diagnosis and earlier surgical treatment the results to be obtained must be incomparably better than in the past.

4. Persistent indigestion in a patient over forty years of age should be taken as a sign that something more than mere indigestion is present, and calls for the fullest, detailed investigation.

5. It behoves the members of the medical profession, therefore, to use every means at their disposal to perfect their diagnosis so that this malady may be recognized early and operation undertaken before it is too late.

Acknowledgements.

In compiling this paper I beg to acknowledge the valuable help received from the Government Statistician (Mr. T. Waites) in supplying the necessary data for Tables I, II and III, and Figure I; the resident medical staff of the State Hospital in compiling abstracts from the hospital register; and from Dr. Eva A. Shipton, honorary pathologist to the New South Wales Community Hospital, who has verified the accuracy of the clinical diagnoses and who will now demonstrate the resected specimens.

INJURIES AFFECTING HEARING.[1]

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INJURIES affecting hearing may be grouped accordingly as they affect the conducting apparatus, the inner ear (cochlea and semicircular canals), the brain.

INJURIES TO THE CONDUCTING APPARATUS.

Injuries to the Auricle.

Injuries to the auricle do not affect the hearing unless a secondary atresia occurs. It is possible

[1] Read at a meeting of Western Australian Branch of the British Medical Association on November 16, 1932.

that the total loss of an auricle may diminish the efficiency of the collecting of sound waves.

Injuries to the External Canal.

Injuries to the external canal cause obstruction by secondary atresia. There are two types of atresia of the external canal: (*a*) partial, that is, affecting the membranous part; or (*b*) total, affecting the membranous and bony segments.

Atresia may be produced mechanically, chemically and physically.

Mechanical atresia may be the result of: (i) Direct injury, such as (*a*) sword cut, as in duelling; (*b*) other injuries; (*c*) post-operative, for example, following a radical mastoid operation. (ii) Indirect injury, as in some fractures of the base.

Chemical atresia may be caused by acids. Throwing acids was a method of personal revenge on the Continent at one time. In the latter stages of the war in Central Europe it occurred as a self-inflicted injury with a view to avoiding service. The accidental instillation of an acid into the canal instead of prescribed drops, for example, pure carbolic acid, may also result in atresia.

Physical atresia may be caused by: (*a*) burns by fire or electricity, (*b*) scalds.

Treatment.—In total atresia a partial radical operation will be necessary. In partial atresia a tube graft from the skin over the mastoid may be utilized to repair the membranous canal if other methods are unsatisfactory. The scar tissue is then excised completely. Hearing then generally returns to normal.

Injuries to the Membrana Tympani.

An injury to the *membrana tympani* does not necessarily interfere with hearing. Damage to the ossicles or adhesions or *otitis media* complicating the injury is a more frequent cause of deafness.

Direct Injury to the Membrana Tympani.

A sharp instrument may pierce the *membrana tympani*, as when wax or a foreign body is being removed.

An impacted foreign body in the ear of a child should be removed under general anæsthesia. If there is considerable trauma in the canal due to previous unsuccessful removal, then wait till the inflammatory œdema subsides before making a further attempt.

A child was sent to the Children's Hospital with an injury to the left *membrana tympani* following difficult removal of a foreign body in the country. The child had acute suppurative *otitis media* on the left side. This resulted in acute mastoiditis, acute tonsillitis (secondary from the ear) and acute mastoiditis of the opposite side.

Organisms introduced into the middle ear through the external canal are dangerous, as they arrive on a mucous membrane which is really a specialized periosteum. This is a poor tissue in which to develop a resistance to cope with such an invasion.

An infection from the naso-pharynx arrives from an area adequately provided with lymphoid tissue, and therefore is generally less destructive and dangerous. Thus a primary invasion from without is more dangerous than a secondary from within.

Other articles may piece the *membrana tympani*, such as hair pins, pens *et cetera*.

Air pressure may cause injury to the *membrana tympani*.

Positive pressure as a cause of injury is seen, for example, in a blow on the ear with the flat of the hand. The rupture is usually on the left side, but in Vienna is on the opposite side, since the back of the hand is generally used. A sudden positive pressure is set up which is equal to the area of the outlet of the ear multiplied by the area of the *membrana tympani* multiplied by the force used. The same mechanics are used to advantage when you thump the bottom of a whisky bottle to remove a cork which has defied a firm pull with a corkscrew.

Positive pressure again may cause injury in high diving, or the sudden force of a breaker in surfing may be the cause. Positive pressure may cause injury in excessive Eustachian or Politzer inflation.

Negative pressure may cause injury, as in kissing on the ear (another Continental custom).

Negative or positive pressure may occur in an explosion (see Figure I). It has been generally accepted that it is the positive pressure which bursts the *membrana tympani*, but certain evidence makes the theory possible that the sudden vacuum following the explosion may be the cause.

FIGURE I.

Photomicrograph (after Fraser). High explosive shell injury of the ear. High explosive shell burst in the room in which this soldier was taking cover. He was semiconscious and had marked deafness. Note slight hæmorrhage in middle ear spaces and in fundus of internal meatus. The labyrinth is normal. Horizontal section showing: 1 and 2, everted edges of rupture in drumhead; 3, handle of malleus; 4, facial nerve with stapedius to left, the *sinus tympani* lies below; 5, posterior canal; 6, utricle; 7 internal meatus; 8, carotid canal; 9, opening of Eustachian tube.

An offensive discharge from an ear generally indicates either a foreign body or a chronic *otitis media*. The only exception to this rule was during the war, when a man, whose *membrana tympani* was shattered by a shell burst, fell into a muddy shell hole. The resulting acute *otitis media* complicating these cases was often offensive from the very beginning.

Indirect Injury to the Membrana Tympani.

Indirect injuries to the *membrana tympani* are those associated with fractures of the skull.

The otoscopic findings of a ruptured drum are: (i) triangular or oval perforation in the lower portion, limited by the radial and circular fibres; (ii) a hæmorrhagic edge to the perforation; (iii) a pale yellow mucous membrane of the middle ear showing through the perforation.

Symptoms of Injury to the Membrana Tympani.

The symptoms of injury to the *membrana tympani* are: pain, tinnitus and some deafness. There may also be faintness, vertigo and nausea.

Treatment.

Do nothing. Powder or drops may produce an acute otitis. Lightly pack the canal with a dry sterile strip of gauze. Syringeing is prohibited.

The Prognosis.

Prognosis is good for simple ruptures, healing taking place in three to six weeks.

The jagged edged ruptures caused by an explosion may leave a permanent perforation due to a turning-in of the edge. Providing there is no inner ear damage, hearing generally returns to normal. Secondary infections of these traumatic perforations always have a doubtful prognosis as regards hearing.

Injuries to the Conducting Mechanism in the Middle Ear.

The conducting mechanism in the middle ear may be injured by: (*a*) fractures (discussed later); (*b*) direct injuries through the *membrana tympani*. In this group the hearing is affected according to the nature of the damage. The percentage of permanent loss of hearing finally depends on secondary infection or a chronic adhesive process. The latter may prove to be progressive up to two years or longer.

INJURIES AFFECTING THE INNER EAR.

Injuries Caused by Sharp Objects.

Sharp objects may penetrate the *membrana tympani* and injure the labyrinth.

Symptoms.—Deafness, vertigo and nausea appear at once. Other symptoms are nystagmus, disturbance of equilibrium *et cetera*. Facial paralysis may be an immediate or late symptom.

Treatment.—The treatment is the same as for injury to the *membrana tympani*, except when labyrinthitis is present, then treat accordingly.

Prognosis.—The prognosis is according to the extent of the injury. If acute labyrinthitis is a complication, then deafness may be total and permanent. If vertigo is due to a total destruction of the labyrinth from secondary infection, then compensation may result in anything up to two years. If the destruction is incomplete, vertigo is likely to be more troublesome.

Transverse Fracture of the Temporal Bone.

Transverse fracture of the temporal bone may affect the inner ear; it will be discussed later.

Concussion Deafness.

Concussion deafness may cause injury to the inner ear. It may result: (i) from an injury to the head (this will be discussed later), or (ii) from an explosion.

If the explosion is powerful, the individual may drop to the ground as if shot and remain unconscious for several hours. On recovery such a patient may be entirely deaf.

If the explosion is less severe, the patient may be dazed for a short time, with some tinnitus and deafness, which generally recover later. When the *membrana tympani* is ruptured, it may save the labyrinth to some extent. The tension is thereby relieved instead of becoming concentrated on the foot-plate of the stapes.

In the differential diagnosis of "explosion deafness" we must consider chronic catarrhal or suppurative *otitis media*, inner ear deafness, syphilitic deafness, chronic adhesive process, otosclerosis and shell shock, or any combination of these. A complete examination and a careful history are important. A history of rupture of the *membrana tympani*, with or without subsequent otorrhœa, is diagnostic, especially if accompanied by a history of bleeding at the time of injury.

Severe explosion deafness with no evidence of interference with the state of consciousness at the time is of very doubtful occurrence. Deafness with total or partial loss of consciousness at the time of a shell burst is very much in favour of injury to the cochlea.

Investigations of patients supposedly suffering from deafness due to shell explosion generally prove the condition to be due to or aggravated by shell shock.

The pathology is believed to be a molecular change in the membranous labyrinth, leading to a degenerative neuritis of the nerve endings, ganglia *et cetera*.

The cochlea suffers less than the vestibular apparatus. Hæmorrhages are generally absent, if there is no rupture of the *membrana tympani*, that is, if the explosion is not close.

The prognosis for these cases is not good if the hearing does not recover soon after the explosion.

Occupational Deafness.

The inner ear is protected by muscles. A tone arriving into the ear produces a reflex contraction of the stapedius muscle, thus fixing the stapes in the window. When the tone is loud, the *tensor tympani* muscle contracts reflexly, thus "fixing" the whole ossicular chain. The ear is thus not easily damaged when exposed to violent sound waves.

This is seen experimentally in animals under narcosis; they are more readily rendered deaf by severe noises, since there is no tension in the tendons of the ear muscles.

It is still a question as to whether air conduction or bony conduction causes more damage. It is better to protect against both of these by wearing cotton wool in the ear and rubber soles on the feet.

The terminal filaments of the labyrinth are subjected to continual irritation by persistent marked

waves in the fluid. These filaments may thus undergo degenerative changes which can result in atrophy. The degeneration begins in Corti's organ and later affects the spiral ganglia and nerve. Wittmaack stated that the degeneration was primary in the nerve and secondary in Corti's organ.

There is also a constant strain in the endeavour to hear above the noise.

The middle ear muscles are often affected, especially the *tensor tympani*. A type of fatty degeneration is set up with loss of efficiency.

Deafness increases gradually. Tinnitus is generally absent. It is very important in people exposed to the danger of occupational deafness to maintain a healthy nose and throat.

Disease affecting the function of the ear deprives it of the necessary protection against this condition.

Caisson Disease.

Caisson workers are liable to deafness if compression or decompression is carried out too rapidly. The deafness is rarely permanent.

Injuries Affecting the Brain.

Injuries affecting the basal nuclei will be discussed later.

Cortical injuries may affect hearing. These patients generally do not survive.

We will now deal with head injuries separately. Let us subdivide head injuries into: (*a*) concussion; (*b*) concussion with ear symptoms; (*c*) concussion of the inner ear; (*d*) concussion of the brain with fracture of the temporal bone: (i) longitudinal fractures, (ii) transverse fractures, (iii) fractures of the tip; (*e*) fracture of the skull combined with suppurative *otitis media*.

Concussion.

The symptoms of concussion are the result of compression within the skull. In this we have three factors: (i) substance compression, (ii) vasomotor paralysis, (iii) fluid compression.

The ganglion cells are actually compressed, and this is called "substance compression".

Vasomotor paralysis results, causing: (*a*) the blood to run more slowly through dilated blood vessels, (*b*) exudate and lymphocytes to leave blood vessels. This so-called "prestatic blood circulation" can disappear very soon after the injury, but it can stay and be permanent. When it remains, it is then part of the pathology of concussion neurosis. Here we have symptoms of headache, change in character (patients easily lose their temper), loss of memory, no resistance against sunshine or alcohol, heart palpitation and inability to work.

In fluid compression the two lateral ventricles are compressed and the fluid is forced through the foramen of Munro into the third ventricle and from the third ventricle into the Sylvian aqueduct and thence to the fourth ventricle. The floor of the fourth ventricle, in which are superficially placed the vestibular and cochlear nuclei, thus shares in the "compression trauma".

Following on concussion of the brain there are always degenerative changes, such as degeneration of nerve cells and glial proliferation. These degenerative changes can be localized in various places. In 40% of cases they are localized in the vestibular and cochlear nuclei.

Concussion of the Brain with Ear Symptoms.

Concussion of the brain with ear symptoms can occur with the sense organ remaining completely normal. The following points should be noted in the symptoms of this condition. Tinnitus is only occasionally present. Hearing is completely normal. Dizziness is present, but has special characters; it is of labyrinthine character, it is slight, it occurs in attacks and is not continually present. Nystagmus is frequently present. It is slight either to one or both sides, and is intermittent. If nystagmus is of the second or third degree, then the case is more than one of concussion. Disturbances in equilibrium may be slight or not present at all. They are very often functionally increased. The head movements test sometimes gives a positive result. The caloric test gives a normal result or reveals hyperirritability. The turning test gives a normal result or reveals hyperirritability.

The prognosis of concussion of the brain with ear symptoms is not good as far as the dizziness is concerned. It tends to increase in intensity with age, and the "free intervals" become shorter.

For the purpose of compensation, how can you tell if a patient suffers from dizziness? If a patient claims he is dizzy and he has spontaneous nystagmus, then he is dizzy. If his caloric test reveals hyperirritability or the result of his head movements test is positive, then the patient has a right to maintain his statement. If these results are not positive, then you cannot confirm the patient's statement. These patients with dizziness have about 40% to 60% loss of efficiency, since they cannot do full work, they cannot leave the ground, they are nervous in thinking they will become dizzy, the prognosis is bad, and we have no certain treatment.

Concussion of the Inner Ear.

Concussion of the inner ear is never present alone in these cases. It is always associated with concussion of the brain.

Of the three factors in brain compression previously referred to, the one which can affect the inner ear is the vascular change. The internal auditory artery, arising from the basilar or cerebellar artery, is affected by this prestatic condition, and a vasomotor internal otitis is set up with emigration of lymphocytes and exudate. Recent researches by Wittmaack indicate that in certain head injuries damage is also inflicted on the organ of Corti in all or most of the coils.

The end organ of the sacculus may be similarly affected. This can occur independently of blood effusion within the inner ear. The cause is probably a sudden compression of the protoplasmic content of the organ of Corti between the fluid on

either side. These two lots of fluid have not had time to readjust their altered pressures.

Diagnosis of Concussion of the Inner Ear.—Symptoms differ according to the situation of exudates. Tinnitus is rare. Hearing is always disturbed, as a rule on one side more than the other, or only one side. Patients are never completely deaf. Use of tuning forks reveals deafness of inner ear type or of the combined middle and inner ear type. Spontaneous labyrinth symptoms are always present, but are slight, that is, slight nystagmus and slight dizziness. The caloric test gives a normal result or reveals hyperirritability, or the response is absent. The result of the turning test is positive, even when the caloric test does not give a positive response.

Prognosis.—The prognosis is not good as far as dizziness or hearing is concerned.

Treatment.—Lumbar punctures are successful in some cases. Of all drugs, calcium and the iodides give the best results.

Concussion of the Brain with Fracture of the Temporal Bone.

The temporal bone is involved in 30% of cases with fractured bases. There are three types: (*a*) longitudinal, (*b*) transverse, (*c*) rupture of the tip. Longitudinal and transverse fractures are of interest only as rupture of the tip is rapidly fatal.

Longitudinal Fracture.

Longitudinal fracture begins in the roof of the mastoid process, along the *tegmen tympani*, into the roof of the Eustachian tube. The line of weakness of the base is through both petro-tympanic fissures, Eustachian tubes, *foramina lacera* and sphenoid. It is therefore a middle ear fracture.

It is often combined with a fracture in the middle and anterior fossæ; more seldom with a fracture in the posterior fossa.

If branches are present in this linear fracture, they are practically always laterally situated, either in the mastoid process or in the superior wall of the bony part of the external canal. Rarely do they go medially into the inner ear.

Typical Changes Found in the Ear in Longitudinal Fractures.—There may be redness in the postero-superior wall of the canal. The *membrana tympani* is very often ruptured; then there is blood in the external ear. The middle ear is more or less filled with blood. The muscles of the middle ear are lacerated, especially the *tensor tympani*. In the majority of cases the ossicular chain is untouched. In a small number of cases there is subluxation of the incus. Changes may occur in the inner ear. In longitudinal fracture the inner ear is normal or has a vasomotor internal otitis, or may have an injury to the sacculus or to Corti's organ from the concussion effects. Petechial hæmorrhages occur only in those cases in which death occurs very soon after the injury. There is never complete deafness. Partial deafness occurs in about 40% of cases.

Recovery.—The fracture heals with connective tissue, never by bone union.

The Diagnosis.—Diagnosis is sometimes impossible with certainty. Of all fractures of the base of the skull, longitudinal fractures of the temporal bone are the most frequent. Many must occur which are only microscopical.

Signs and Symptoms.—Bleeding from the ear is not a certain sign, since external injuries may cause this. Be careful the blood has not gained entrance from a cut scalp. If hæmorrhage is profuse, then it comes from the middle meningeal artery or the sinus, and should be dealt with accordingly. If there is cerebro-spinal fluid from the external canal, then this is diagnostic of a longitudinal fracture with rupture of the dura. In such cases the treatment is to put in a sterile dressing and change it frequently. Do not attempt to syringe or clean out the canal.

In regard to findings in the external canal, one can very often find a redness in the postero-superior wall of the external canal. This is not diagnostic of longitudinal fractures, but can happen after a number of other injuries. If a "step" appears in the external canal, this is significant.

In regard to findings on the *membrana tympani*, a rupture or a hæmatotympanum will be present. In the latter case the *membrana tympani* looks bluish-black. Be particular not to interfere with the external canal in any way during your aural examination. It is criminal to examine with a probe.

When functional tests are carried out, 30% or 40% show a marked diminution of hearing.

X ray examination is reliable only when the findings are definitely positive.

Prognosis.—As far as life is concerned, the prognosis is always doubtful. The patient may later develop an *otitis media* and succumb to meningitis or brain abscess.

Hearing.—Any deafness remaining after two months has a poor prognosis, since it diminishes as time goes on. This is due to organization of the blood clot and a consequent chronic adhesive process. Nevertheless, careful treatment of the adhesive process may lead to some improvement of hearing.

Transverse Fractures of the Temporal Bone.

Transverse fractures of the temporal bone cross, as a rule, in the region of the jugular bulb, across the pyramid, and end on the anterior surface of the pyramid. They are frequently combined with fractures of the posterior fossa. The fracture is typically one affecting the inner ear.

Signs and Symptoms.—Signs and symptoms are illustrated in the cases depicted in Figures II and III.

The vestibulum is pressed upon and badly damaged.

The cochlea and semicircular canal are destroyed more by bleeding than by fracture. Fibrous tissue

later forms in these spaces, and as a late result the organ of Corti and the eighth nerve ganglia atrophy. The labyrinth may be indirectly injured by hæmorrhage travelling along the eighth nerve.

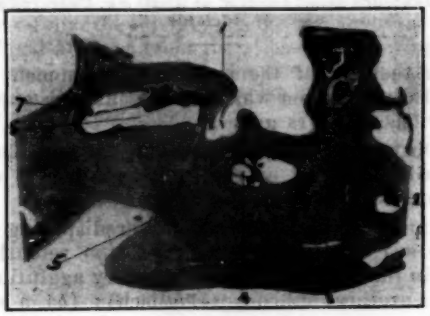

FIGURE II.

Photomicrograph (after Fraser). Man fell from a scaffold nine feet high. Was conscious, but blood and cerebro-spinal fluid flowed from right ear. Patient was very deaf. Later, convulsions and conjugate deviation to the side of the labyrinth lesion occurred. Patient was operated on, but died. Horizontal section through right ear in region of round window. 1, line of fracture; 2, blood in convexity of posterior canal; 3, endolymphatic aqueduct free from hæmorrhage; 4, ampulla of posterior canal with blood in it; 5, inner ear of fracture reaches internal auditory meatus; 6, *scala tympani* of basal coil with hæmorrhage; 7, cochlear canal contains blood.

The middle ear is affected when the fracture of the labyrinth connects the two windows.

In a very small percentage of cases the inner ear is not affected at all.

The sinus may be injured in 1% of cases.

FIGURE III.

Photomicrograph (after Fraser). Fracture of cranial base with injury to labyrinth capsule. Horizontal section through right ear just above round window. 1, fracture of promontory; 2, both peri- and endolymph spaces of posterior canal contain blood; 3, vestibule with slight hæmorrhage; 4, line of fracture reaches canal for nerve to ampulla of posterior semicircular canal; 5, basal coil of cochlea with hæmorrhage in all three scalæ.

The seventh and eighth nerves may be injured after their entrance into the auditory canal. If signs of their involvement occur late, they are probably due to hæmorrhage, and recovery will be more rapid. It is not usual for patients with this type of injury to survive, except, perhaps, children.

The fracture heals only by connective tissue; bony proliferation may take place in the labyrinth subsequently.

Diagnosis.—On X ray examination the transverse fracture is more gaping; therefore, a positive finding is more frequent than in the longitudinal type. It is possible also to get a positive X ray finding some time after the fracture has occurred, as there is no bony union.

Functional tests reveal complete deafness and signs of a destroyed labyrinth. The *membrana tympani* is generally normal. Thus deafness with a ruptured *membrana tympani* is of better prognosis in a fractured skull than deafness with no rupture of the *membrana tympani.*

Fracture of the Skull Combined with Suppurative Otitis Media.

German otologists hold that one should operate prophylactically. Brunner, of Vienna, reports seventeen cases of middle ear suppuration with fracture of the temporal bone in which recovery occurred without operative interference.

Patients with bony suppuration, especially cholesteatoma, generally require operation, but those with middle ear suppuration should be treated conservatively.

Patients with fracture who acquire a suppurative *otitis media* should have an early and free incision of the *membrana tympani.* If the symptoms then subside, no more is necessary. If the symptoms progress, then you must operate. Meningitis, brain abscess, extradural abscess and sinus thrombosis are the complications to be feared.

If you operate, expose the whole of the fracture. The type of operation will be a cortical or radical mastoidectomy or a labyrinthectomy, according to the findings. Aim at free drainage and no retention. It is advisable to avoid jarring by hammer or chisel. One must, of course, consider whether the state of the patient will permit of radical treatment.

Prognosis.—The prognosis for life is worse in transverse fractures than in longitudinal fractures, owing to the injury affecting structures more intimately connected with the brain. About 15% of patients die. In 8% of "skull fracture" death results from meningitis caused by infection from the naso-pharynx. Of this meningitis, 92% of cases are due to acute increase in brain pressure or brain laceration or both.

If the patient survives forty-eight hours, the prognosis is more favourable.

The prognosis for the function of hearing is hopeless in transverse fractures. If a bilateral injury of this nature occurs to a child below the age of seven, then deaf mutism will result (see Figure IV).

Treatment of Fractures of the Temporal Bone.

A patient with a head injury should have an examination of the ear (and its function) as soon after the accident as possible. This is important subsequently for purposes of compensation. Any patient with an injury affecting the ear should be carefully watched. The early diagnosis of *otitis media* is essential, since early surgical intervention in the complications of these cases will give the best chance of recovery.

FIGURE IV.

Photomicrograph (after Fraser). Deaf-mutism due to fracture of skull in childhood. × 6 diam. 1, footplate of stapes; 2, perilymphatic space of vestibule largely filled up with new formed bone; 3, dilated saccule; 4, cochlea: the basal coil especially shows new formed bone and connective tissue; 5, markedly atrophic eighth nerve. From a male deaf-mute, aged sixty-four, who, at the age of four, received severe head injury.

Conservative Treatment.—Conservative treatment consists of cold compresses to the shaven skull, intravenously administered hypertonic saline solution and hypertonic glucose solution, repeated lumbar punctures, light diet, sedatives, stimulants, such as caffeine *et cetera*.

Surgical Treatment.—As far as primary primary decompression is concerned, 8% of deaths from fractured bases are due to meningitis; 92% of deaths are due to increased brain pressure. From these statistics it would appear that a decompression is generally indicated when signs of compression persist in spite of repeated lumbar punctures. If done at all, it should be done early. At operation the fracture is exposed and enlarged. When it cannot be found, a subtemporal decompression should be done. Further shock may be avoided by the use of local anæsthesia.

ACKNOWLEDGEMENTS.

I wish to acknowledge the teachings of Brunner, Ruttin, and Fraser, from which I have made references.

THE MODE OF DEVELOPMENT OF A VESICAL DIVERTICULUM.

By W. JOHN CLOSE, M.S. (Adelaide), F.R.C.S. (Edinburgh),
Honorary Demonstrator of Anatomy, University of Adelaide.

THE theories of the mode of development of a vesical diverticulum which at present gain approval in various quarters are:

1. Obstruction to the emptying of the bladder, generally conceded as the exciting cause in either the congenital or acquired types.

2. Intrauterine conditions, the predisposing cause of the so-called congenital types: (*a*) retention of urine in the fœtus, due to a temporary agglutination of the urethral mucosa (Englisch); (*b*) a superabundance of embryonic tissue in the bladder wall (Pagenstecher, cited by Young); (*c*) the formation of an excess of epithelial tissue at the fusing edges of both Wolffian and allantoic elements of the bladder, and a temporary failure of epithelialization between the two (Watson, cited by Lower); (*d*) supernumerary ureteric buds (Swift Joly); (*e*) patent urachus, the probable origin of all cases found at the vertex.

This ætiological *résumé* has been abstracted from Young's "Practice of Urology", 1926,[3] and "Cabot's Modern Urology" (Lower), 1926,[2] their references being unobtainable, and therefore not open to criticism here.

The general opinion is that congenital diverticula do occur, but Young, quoting Hinman, writes: "the majority of congenital diverticula in infants and children usually show signs of obstruction", so that all types can be considered to require an obstructive factor for their formation, and until this takes place the diverticulum is seldom in evidence. Hence we rarely find them till the fourth decade, and the greater percentage not till the sixth.

I am about to draw attention to another condition, which is also a developmental one, and which may be the predisposing factor in either congenital or acquired types. Pulsion from within is a palpably unconvincing reason by itself, when one finds diverticula in a bladder which has been submitted to only a mild interference, and less convincing when the most one sees in the majority of severe obstructions is a number of shallow saccules. There is no need to postulate a congenital muscular deficiency, since this is apparent cystoscopically, even in the small saccules. A series of dissections made by me in the Adelaide University Medical School has suggested that traction from without is just as important as pulsion from within. The œsophagus provides us with an analogy in both types of diverticula, the traction type caused by the scar tissue of a tuberculous lymphadenitis at the tracheal bifurcation, and the pulsion type at the lower end of the pharynx.

One must understand the nature of the endopelvic fascia to visualize the importance of the allantoic

sheath of Delbet,[1] that condensation of mesoderm which surrounds the bladder and urachus, extending upwards with the urachus from the bladder apex, and laterally from the lateral margins of the empty bladder towards the obliterated umbilical artery. The development of this lateral sheet of fascia has occurred by the divergence in the embryo of the umbilical arteries from the allantois, carrying away with them a process of the allantoic sheath from the whole of the allantois (see Figure I, A, B and C).

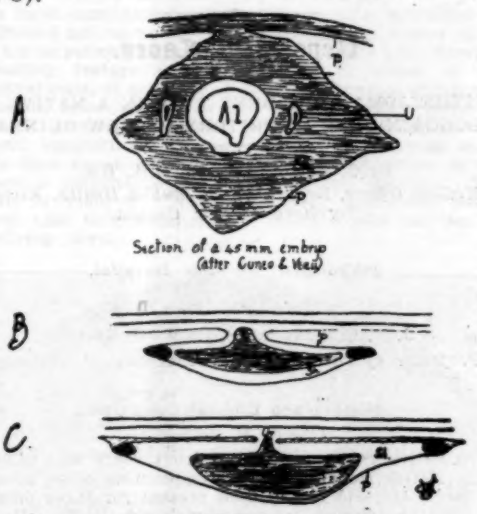

FIGURE I.

Showing mode of development of the allantoic sheath. Al. = allantois, U. = umbilical artery, Sh. = allantoic sheath, P. = peritoneal covering, Ur. = urachus.

In A the contents of the sheath are seen to be completely surrounded by peritoneum, with an antero-lateral *cul-de-sac* on each side.

In B the umbilical arteries are diverging, and in C two layers of peritoneum have fused to form the umbilico-prevesical aponeurosis of Charpy, the bladder and urachus having become completely extraperitoneal.

In the filling adult bladder the slack of this process is gradually taken up by its expanding walls, until these lie in contact with the lateral pelvic walls, when the whole of the allantoic sheath is utilized in the envelopment of the bladder, the obliterated umbilical arteries lying in direct contact with the viscus.

Frequently in my dissections I have found a projection of this sheath into the obturator foramen. A photograph of such a condition is supplied in Figure II, where a mammilliform process has been released from the foramen.

I concluded at first that this was a rare condition, since the only anatomist mentioning it (Delbet) said that he had once observed it. I saw it, however, in three of the first five pelves dissected, and in each of the three a considerable bladder diverticulum was found on the attached side, the apex of the mucosal protrusion being held firmly by such an attachment. I have, however, found such an endofascial attachment on several occasions since then, without any diverticulum.

It seemed then that pulsion and traction were each necessary to obtain the bladder protrusion, but in my formalinized subjects it was impossible to say whether any obstruction had been present, and

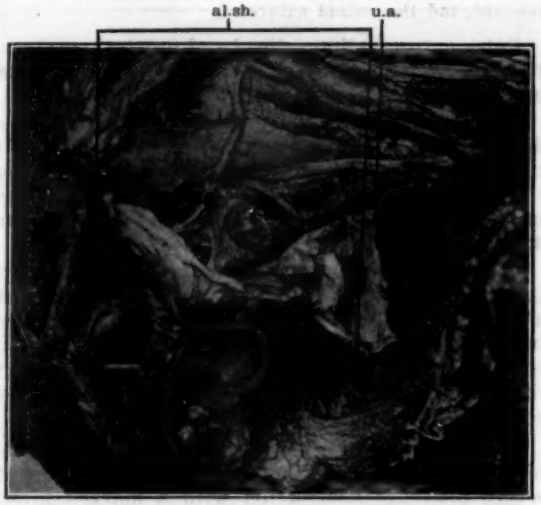

FIGURE II.

A dissection of the endopelvic fascia of a female pelvis, sagittally bisected. al.sh. = the cut edge of the allantoic sheath, a triangular portion above this level having been removed, and the remainder drawn medially to show the mammilliform projection near its lateral (in the displaced condition shown by the photograph, its posterior) extremity. u.a. = the umbilical artery, that is, in this extent the superior vesical artery, foreshortened in the photograph, limiting a lamina of fascia which it has raised from the common neuro-vascular sheath, which lamina becomes continuous with the allantoic sheath anteriorly.

it was necessary to wait for an operation case to demonstrate the presence of both factors, and the following is a synopsis of a case which showed them.

A male, aged forty-two years, complained, on June 19, 1931, of a gleety discharge, cloudy urine with threads, and frequently very thick masses of purulent material. He had been treated for three to four years for "stricture and prostatitis". Occasional bouts of frequency occurred, but when seen he was normal in this respect.

He had gonorrhœa eighteen years ago, and six years ago had a discharge which cleared up without the intervention of a doctor, and which presented no acute signs. He had a swollen testicle three to four years ago.

On examination a muco-purulent urethral discharge was present, films showing a multiplicity of flora, but no gonococci. (Culture of the prostato-vesicular secretion at a later date failed to grow the gonococci.) The urine was thick, with many filaments and *débris*. On rectal examination thickening round the vesicles was felt on both sides.

Anterior urethroscopy revealed hard infiltration of the greater part of the penile urethra, so that at first only a number 12 Charrière gum elastic catheter could be introduced comfortably. Dilatation was performed, however, without trauma till a number 30 Charrière instrument was reached in less than three weeks, and a number 32 Charrière for the posterior urethra.

On July 9, 1931, as the three glass test showed all specimens to be cloudy, cystoscopy was undertaken. The bladder was free from cystitis, and trabeculation was slight. The mouth of a diverticulum was seen on the left side, above and lateral to the ureteric orifice, and large flakes were seen emerging from the opening.

Intravenously injected indigo-carmine was excreted from both sides in under five minutes. On July 15, 1931, under spinal analgesia the bladder was opened suprapubically,

the diverticulum was packed with gauze (appearing about the size of a duck's egg), enucleated extravesically from the allantoic sheath, removed, and the mouth sutured intravesically. The lateral extension of the allantoic sheath was followed, and, as expected, was found to be anchored at the obturator foramen. This attachment was released and the wound sutured.

Pulsion, from mild urethral and prostatic obstruction, as well as traction, were thus found in this case.

In Young's series of 41 cases, 78 diverticula were distributed as follows: at the right ureter, 11; at the left ureter, 9; on the right lateral wall, 20; on the left lateral wall, 14; at the base, behind the interureteric bar, 17; at the vertex, 4; and on the anterior wall, 3.

It is thus seen that 54 of the 78 began laterally, though it does not seem necessary to point out that a lateral diverticulum, having once begun, whether influenced by traction or not, need not necessarily continue in the lateral direction. Indeed, many of them take a posterior course, as if the line of least resistance were along the ureteric channel.

What of those commencing behind the base of the bladder, a condition confined entirely to cases of severe prostatic obstruction with a marked interureteric bar, according to Young? I have not seen one commencing here, that was sufficiently large to assume the dimensions of a diverticulum, though the frequency of deep saccules, which empty at the same time as the bladder, are of common observation in prostatics. Assuming that such types do occur, and even rejecting the possibility of the influence of traction from a lateral direction, traction may still come into play from the fibrous tissue associated with a chronic vesiculitis, a condition frequently enough demonstrated *per rectum* in prostatic obstruction.

I have seen one case that was evidently due to fibrous traction other than the developmental condition described above.

I had operated on a patient for multilocular parovarian cysts, which had burrowed rather extensively into the parametrium. At a later date I had occasion to open her abdomen again for the purpose of performing a hysterectomy for fibroids. I was not surprised, on examining her pelvic viscera, to find another cyst embedded in the endopelvic fascia, on the side from which the cysts had previously been removed, but was indeed surprised when, on attempting to shell it out, and perforating it in the process, I found it to contain urine. I had opened a diverticulum which communicated with the bladder by a narrow neck.

This was at a time when I was not interested in the evolution of a diverticulum, but even then it was obvious to me that traction of the scar tissue, which resulted from the reefing of all the involved endopelvic fascia, had been the cause.

Summary.

Traction of fibrous tissue, notably a developmental attachment of the allantoic sheath to the obturator foramen, appears to have a predisposing influence in the formation of many diverticula of the bladder, and, when present, may be capable of confirmation at operation or autopsy.

Acknowledgement.

My thanks are due to Professor H. J. Wilkinson, of the University of Adelaide, for providing me with facilities for anatomical research in the Medical School, and for assistance in many other ways.

References.

(1) Paul Delbet: *"Traité d'Anatomie Humaine"*, Volume V, Part I, Third Edition, 1923, page 168.
(2) W. E. Lower: "Cabot's Modern Urology", Volume II, 1926, page 56.
(3) H. H. Young and D. M. Davis: "Practice of Urology", 1926, Volume I, page 33, and Volume II, page 60.

Reports of Cases.

EPITHELIOMA OF RODENT TYPE IN A NATIVE OF BOUGAINVILLE, TERRITORY OF NEW GUINEA.

By C. MERVYN DELAND, M.B., B.S.,
Medical Officer, Department of Public Health, Kieta, Territory of New Guinea,

L. B. BULL,
Pathologist, Adelaide Hospital,

AND

J. B. CLELAND, M.D., Ch.M. (Sydney),
Professor of Pathology, the University of Adelaide.

History and Clinical Condition.

(By C. M. Deland.)

THE native Tauni, aged about thirty years, was admitted to the Native Hospital, Kieta, complaining of an ulcer on the face. He said it had been present for three months; but such testimonies are notoriously unreliable. He had been treated with "Novarsenobillon" on the supposition that the lesion was a frambœsial one. The ulcer was painless.

On examination he was otherwise healthy. The skin colour was black, with a slight brownish tinge, as is usual in natives of the area. There was an ulcer in the left temporal region, extending from the point of the ear to the orbit, and commencing to involve the latter. It looked a typical rodent ulcer, and was inoperable. There were a few bleeding points (see Figure I).

FIGURE I.

A section was removed from the edge and forwarded to Rabaul for diagnosis. Dr. Backhouse reported: "An epithelioma, somewhat more active than a typical rodent." A second report, from Professor Cleland and Dr. L. B. Bull, of Adelaide, is appended.

Comment.

The interest of this case is in the great rarity of such a growth in a black skin.

Acknowledgement.

I have to thank the Director of Public Health of the Territory of New Guinea for permission to publish this case, and Dr. Backhouse, of Rabaul, and Dr. Bull and Professor Cleland, of Adelaide, for their reports on the section.

Pathological Report.

(By L. B. Bull and J. B. Cleland.)

We have examined the interesting section forwarded by Dr. Deland and agree that the neoplasm is a rodent ulcer. It is not as reticulated as many rodent ulcers are. Another interesting feature is the presence, in many of the epithelial cells, of granules of melanin pigment, which also appear in some of the stroma cells between. Diffusely scattered groups of plasma cells and lymphocytes are present, suggesting that the growth is somewhat more active than usual, or that the extensive ulceration of the surface is responsible for the round cell reaction. The failure to yield to "Novarsenobillon" injections would suggest that this inflammatory reaction was not due to underlying yaws.

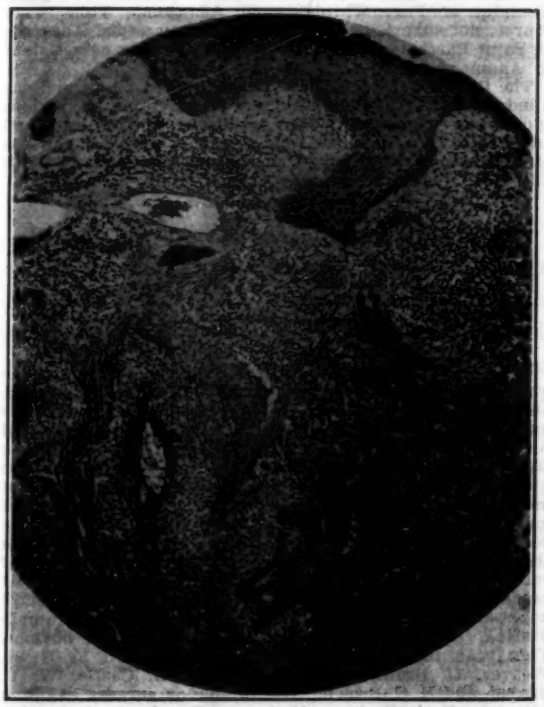

FIGURE II.

Photomicrograph of section of rodent ulcer of the face of a native of Bougainville, Territory of New Guinea (×70).

A photomicrograph has been taken, at Dr. Deland's request, by Mr. G. C. McLennan, which shows clearly the rodent ulcer character of the growth. Dr. Deland's photograph of the patient shows an extensive growth. The age of the patient (about thirty) is an unusual feature. The occurrence of such a growth in a dark-skinned race is of considerable interest, more particularly in view of the suggestions that have been made that these neoplasms are more frequent in persons exposed to the rays of the sun and unprotected by adequate pigment. The presence of melanin in some of the tumour cells suggests that these retain the capacity to deal with melanin pigment that is shown strikingly by the basal layer of the epithelium in these natives. Castellani and Chalmers[(1)] state that rodent ulcer is comparatively rare among natives and that they have seen one case in a Sinhalese woman.

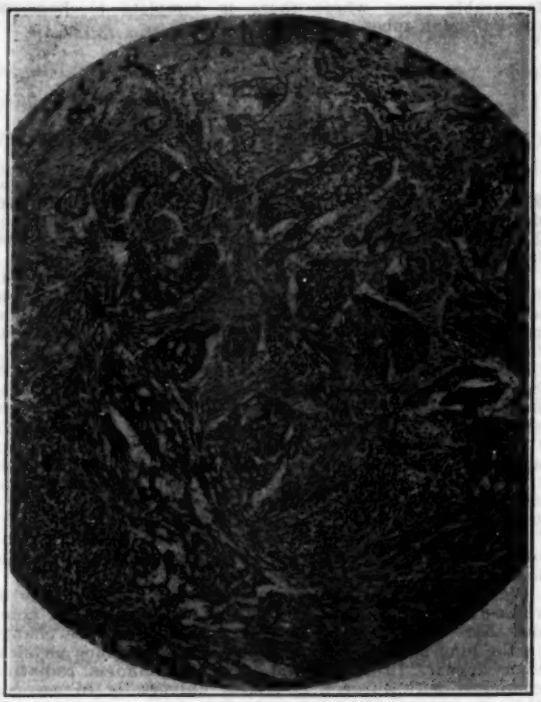

FIGURE III.

Photomicrograph of section of rodent ulcer of the face of a native of Bougainville, Territory of New Guinea (×70).

Reference.

(1) Castellani and Chalmers: "Manual of Tropical Medicine", 1919, page 2277.

SALPINGOSTOMY.

By CECIL COGHLAN, M.B. (Sydney), F.R.C.S. (England), F.R.C.P. (Ireland), F.R.A.C.S., D.G.O.,

Honorary Assistant Gynæcologist, Saint Vincent's Hospital; Honorary Gynæcologist, the New South Wales Community Hospital, Sydney.

IN the issue of THE MEDICAL JOURNAL OF AUSTRALIA of April 30, 1932, on page 608, I published an article on salpingostomy, in which I reported a series of seventeen cases in which I had performed this operation, with three successful pregnancies resulting. This series was complete to December, 1931.

It may be of general interest, and I think that it is certainly worth recording, that, since the date of publication, two further pregnancies have occurred in this series. One patient has conceived and given birth to a second child, and a further patient is now four months pregnant. One patient of this series has lost her husband since the operation was performed, so, for statistical purposes, the series may be considered as sixteen.

A successful conception has thus occurred in 25% of the cases in which operation was done, and I think that it is safe to claim that, without this operation, these children would never have been conceived.

The operation was not done at random, but the cases were chosen from those in which a salpingograph showed that there was a reasonable chance of a successful issue, and where, also, the husband had had a thorough examination.

Such a result as this, I think, definitely places salpingostomy as a procedure to be adopted in certain favourable cases where there is complete blockage of the Fallopian tubes.

Reviews.

THE VALUE OF RADIOLOGY IN MAXIMS.

THIS small handbook of radiologic maxims, by Harold Swanberg, is a collection of most informative material dealing with the value of X rays in diagnosis and of X rays and radium in the treatment of various lesions, both deep and superficial.[1]

The book is of interest to radiological specialists, but it should prove of greater value to the general practitioner and to physicians. Practically all phases of radiology and radiotherapy are dealt with in a concise manner without technical details of exposure or technique.

The author stresses the point that the radiologist must be a qualified medical man with wide general clinical knowledge, and that, unless specially qualified, no man should attempt the wide use of X rays. His remarks that "many physicians in general practice, who own x-ray apparatus, merely dabble, and make a bad mess of what is truly a highly specialised subject" will receive general approval from the specialist, who so often sees ridicule brought on X ray work by these meddlers.

Many famous physicians' views are given on the value of X rays, and such men as Lord Moynihan, Mayo, Crile and Bloodgood are freely quoted.

Various paragraphs draw attention to the accuracy of radio-diagnosis in gastric ulcer and carcinoma, and point out that in cases of pulmonary tuberculosis some change in the lung fields is demonstrable before ordinary physical signs appear. In uterine and cervical carcinoma, radiation combined with surgery is almost universally in use. A list of skin conditions in which radiation is of benefit is also included.

DERMATOLOGY.

DR. ROXBURGH'S book, "Common Skin Diseases", is, in our opinion, the best work of its kind at present available.[2] Without going into detail unnecessary for the purposes of the section of medical readers for whom it is intended, namely, students and general practitioners, the author nevertheless introduces each of the common diseases in the full light of all the recent knowledge of dermatology and general medicine which can be brought to bear upon it.

The "Index of Preliminary Diagnosis" with which the book commences gives the reader a key to the description of eruptions similar to that with which he is confronted and enables him to save much time in the search for the information he requires.

Throughout the classification is based upon ætiology as far as the present day knowledge of causation of individual diseases will permit. Where the ætiology is unknown, as in psoriasis, the disease is described outside the general grouping.

Syphilis in its ætiology, histopathology, course and clinical features is admirably treated, and it is noteworthy that Dr. Roxburgh has eliminated the old confusion in which the student's mind was left under the old method in which papules, condylomata, cutaneous gummata and the rest were described as different lesions, instead of modifications dependent upon the conditions existing at the site of their appearance or upon the age of the infection. The omission of treatment from the account given of syphilis is in some respects regrettable, but we would rather have this description of syphilis without any account of treatment than many others, even when accompanied by the soundest system of therapeutic measures for the disease.

The tumours are described briefly and clearly and in a manner which is, no doubt, adequate for European needs. This, however, is the only section of the book where the student and practitioner in Australia will find the information available inadequate for practice in a country where pre-epitheliomatous keratoses, basal and squamous celled carcinoma form such an important part, not only of dermatology, but of country practice. In the account of the ætiology of skin cancer, the influence of sunburn irritation is explicitly admitted probably for the first time in European text books. This is, to say the least, gratifying to Australian practitioners, who have contended for many years that the only explanation for the disproportionate frequence of skin cancer in Australia as compared with Europe is the intervention of this ætiological factor.

Throughout the book one is ever conscious that Dr. Roxburgh is of the Colcott Fox school, and even the arrangement of the book is reminiscent of the two great disciples of Fox, Whitfield and Adamson. This is, of course, not surprising since Roxburgh succeeded Adamson at Saint Bartholomew's Hospital after serving as assistant to Adamson for many years.

The illustrations consist of eight coloured plates and one hundred and ten photographic reproductions of various types and phases of skin eruptions. They are all admirable. We commend this book not only for the use of students, but for that of general practitioners who will find it an indispensable part of their library equipment.

[1] "Radiologic Maxims", by H. Swanberg, B.Sc., M.D., F.A.C.O., with Foreword by H. Schmitz, A.M., M.D., LL.D., F.A.C.R., F.A.C.S.; 1932. Quincy (Illinois): Radiological Review Publishing Company. Large Crown 8vo., pp. 126. Price: $1.50 net.

[2] "General Practice Series: Common Skin Diseases", by A. C. Roxburgh, M.A., M.D., B.Ch., F.R.C.P.; 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 342, with eight plates in colour and 110 illustrations in the text. Price: 18s. net.

Notes on Books, Current Journals and New Appliances.

PROGRESS IN MEDICINE DURING 1932.

"THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS" is so well known that little more than a notice of the appearance of the several volumes is required.[1] These volumes published in America and the well known "Medical Annual" published in Great Britain are indispensable to the practitioner who wishes to make an attempt to keep himself abreast of progress in the various branches of medicine, without the labour of delving into the maze of medical journals of all the countries of the world. The books of the "Practical Medicine Series", as explained in these columns on previous occasions, are composed of references to articles on different subjects, suitably arranged. When the editor of the section has deemed necessary, he has added a word of explanation or of amplification. The advantages of this arrangement are obvious. This year the volume on medicine is edited by G. H. Weaver, L. Brown, G. R. Minot, W. B. Castle, W. D. Stroud, and R. C. Brown. The volume on surgery has been edited by E. A. Graham; that on pædiatrics by I. A. Abt; that on obstetrics and gynæcology by J. B. de Lee and J. P. Greenhill; that on eye, ear, nose and throat by E. V. L. Brown, L. Botham, G. E. Shambaugh and E. W. Hagens; that on dermatology and syphilology by F. Wise and M. B. Sulzberger; that on neurology and psychiatry by P. Bassoe and F. G. Ebaugh; and that on urology by J. H. Cunningham. The books of the series for this year will doubtless be as useful and popular as they have been in previous years.

[1] "The Practical Medicine Series: Series 1932": "General Medicine", pp. 830; "General Surgery", pp. 816; "Pediatrics", pp. 564; "Obstetrics and Gynecology", pp. 679; "Eye, Ear, Nose and Throat", pp. 686; "Dermatology and Syphilology", pp. 471; "Neurology and Psychiatry", pp. 488; "Urology", pp. 464. 1932. Chicago: The Year Book Publishers. Crown 8vo., with illustrations.

The Medical Journal of Australia

SATURDAY, JUNE 10, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ENTERIC FEVER AND CHRONIC CARRIERS.

DURING the last few decades there has been a steady diminution in the number of deaths occurring in Australia from enteric fever. During the period 1911-1915 there were 2,848 deaths; during the period 1921-1925 there were 1,209 deaths; and during the period 1927-1931 there were 652 deaths. These figures represented a rate per million of population of 119, 43 and 21 respectively. During 1931, 342 infections were notified in New South Wales, 203 were notified in Victoria, 159 in Queensland, 64 in South Australia, 70 in Western Australia, 22 in Tasmania, and none in either the Federal Capital Territory or in Northern Australia. The diminution in the number of cases and in the number of deaths is the result chiefly of improved sanitation and of a good water supply. At the same time there is still an enteric fever problem in the Commonwealth. Because fewer infections are occurring, there may be a tendency to regard them with complacency; particularly is this likely to happen as far as the carrier state is concerned. More than one writer has shown in these pages that the carrier state is sometimes difficult to discover; but, however difficult the discovery may be, the effort is always worth while, for many an outbreak of infection has been suddenly checked with the discovery and adequate treatment of a single carrier.

The Medical Research Council of Great Britain regards the carrier problem in enteric fever as of such importance that it has added to its special reports one on chronic enteric carriers and their treatment.[1] In the preface to the report the Council states that, though the enteric fevers are responsible for only a small part of the morbidity and mortality among the peoples of Great Britain, these diseases are common in other countries with temperate climates. The report has been written by Professor C. H. Browning, of the Department of Pathology of the University and of the Western Infirmary, Glasgow. In his investigations he has had the assistance of Dr. H. L. Coulthard, Dr. R. Cruickshank, Dr. K. J. Guthrie and Dr. R. P. Smith. Though the report is based on certain investigations made by these workers, it contains references to work done in many other centres, and must be looked upon as a reliable summary of present day knowledge of the subject. Obviously no student of preventive medicine, particularly in a country like Australia, which is still faced with the carrier problem, can allow to pass unnoticed what Professor Browning and his colleagues have written. By drawing attention to some of the more important features of the report we hope to demonstrate its value.

The report is concerned chiefly with the chronic or permanent carriers of bacilli of the typhoid-paratyphoid group, who may or may not have presented manifestations of an acute infection, but who continue for long periods to harbour and excrete the specific organisms, usually in fæces or in urine or in both. Any person who, after complete clinical recovery from an attack of enteric fever, continues to discharge the specific bacilli, is regarded as a carrier. A distinction must be made between temporary and permanent carriers. Three statements are made in the report in this regard: (i) In convalescence from enteric fever a consider-

[1] "Chronic Enteric Carriers and their Treatment", by C. H. Browning, with H. L. Coulthard, R. Cruickshank, K. J. Guthrie and R. P. Smith. Medical Research Council of the Privy Council, Special Report Series, Number 179, 1933. London: His Majesty's Stationery Office. Pp. 80. Price: 1s. 6d.

able number of persons excrete the specific organisms for short periods; these are temporary carriers. (ii) The majority of those who continue to excrete enteric bacilli for six months after the attack, will continue to do so. (iii) Those who continue to excrete the bacilli after a year, will not become cured spontaneously and are to be regarded as permanent or chronic carriers. The identification of carriers depends on the isolation of the specific organism from discharges and secretions. Since this is the special work of the bacteriologist, no further reference will be made to it. As far as chronic urinary carriers are concerned, it is concluded that the frequent presence of gross lesions in the urinary tract is sufficient to account for the failure of treatment by antiseptics and vaccines. Intestinal carriers present a more difficult problem. It has been found that drug treatment, physiotherapy, measures aimed at the alteration of the flora of the intestine, and vaccine therapy have proved ineffective. The most satisfactory results have been obtained with surgical operations on the biliary tract. Professor Browning and his colleagues quote in detail three cases in which gall-bladder operations were effective. They also state that, according to collected records, cholecystectomy has been effective in 75% of cases. Unfortunately, in a certain proportion of intestinal carriers, operation on the gall-bladder will not effect a cure, because the specific bacilli may have their sole habitat in the intestine, and apparently there is no way of distinguishing these carriers before operation. The significant statement is made that Lyon's biliary drainage has not been sufficiently tested. This should be done, for it is not right to subject to a serious surgical operation a person who is not obviously ill if there is some possibility of his being cured by treatment with the duodenal tube.

Any discussion on the carrier state would not be complete without reference to its prevention. In this connexion emphasis is laid on adequate care of the patient during the acute attack and during convalescence; this includes the giving of a diet with a high caloric value. Some observers hold that those who contract the disease in spite of having received enteric vaccines, are less liable to become chronic carriers; there is some evidence to support this view. Vaccination of the general population against enteric infections is scarcely practicable, but too much stress cannot be laid on the necessity for vaccination of the staffs of hospitals and of patients in certain institutions, such as hospitals for the insane and hospitals for infectious diseases. The problem would be largely solved if every patient known to have suffered from an enteric infection were submitted to searching bacteriological examination at intervals of two or three months after he was clinically cured, and if a diligent search were made for the source of every known infection. This will not happen until every general practitioner regards investigations of this type as part of his routine duty.

Current Comment.

EMPHYSEMA.

EMPHYSEMA is one of the pathological states that exemplify the truth that no subject is played out. Morphologists are daily discovering that the ordinary descriptive anatomy, acquired with such pains during the days of studenthood, and lost only too easily, is by no means always correct, as used to be believed. Morbid anatomy, even of the macroscopic variety, is ever fresh also, and the pathologist who, like some scientific Paul of Tarsus, sets out to take the advice he gives, of proving all things, will find his subject still fresh and interesting. Emphysema is one of the neglected subjects. Clinicians look at the thorax of the elderly man and often without hesitation pronounce it "emphysematous" because it is "barrel-shaped" and fixed. The true correlation of the external configuration of the bony cage of the chest with the pathological state (if any) of the organs contained therein is not always attained. Not infrequently it happens that radiological study of the chest shows that there is no evidence of gross emphysema, but that calcification of the costal cartilages and other degenerative changes have altered the mean position of the bellows. The distinction drawn between emphysema of the atrophic and hypertrophic kinds should also be remembered.

W. B. Kountz and H. L. Alexander have recently written on the subject of non-obstructive emphysema.[1] They remark that the origin of the condition is usually regarded as being in the lungs, but contend that the mechanics of the cage must be taken into account also. They do not support the statement of Freund that the cause of non-

[1] *The Journal of the American Medical Association*, February 25, 1933.

obstructive emphysema is ossification of the costal cartilages and ankylosis of the costo-vertebral junctions. Instead, they claim that radiological and, in particular, histological studies favour the site of the causal lesion as being in the intervertebral disks. Pathological evidence shows that the nuclei of the disks soften, swell and degenerate. Lime salts are laid down, and the degenerative change invades the vertebral bodies, which become thinned and "invaded". Collapse follows, bringing about kyphosis, with consequent alteration in the conformation of the chest. The thoracic cage is increased in its lateral diameter, though the vertical diameter is shortened. However, the authors state that while with the large-lunged condition of chronic emphysema there is impairment of respiratory capacity, when the lungs are little increased in size there is *per se* but little loss of respiratory function. All this depends on a morbid anatomical refinement which, at the present time, does not appear to have any definite practical application, but one very important point emerges. We should be extremely careful in saying that a patient's chest is emphysematous. Sometimes determination is easy. The young sufferer from chronic asthma and relapsing bronchitis can hardly be the subject of degenerative, or at least senescent, change. But the problem is different in the middle-aged and elderly. It should therefore be remembered that when a patient is encountered who has a chest that is fixed and altered in the ratio of its lateral and vertical diameters, there are three possibilities. He may have an emphysema of pulmonary origin, he may have emphysema of extra-pulmonary origin, or he may merely have a fixed chest with no demonstrable change in the lungs, or at least a change that is inconsiderable. The possible importance of correct thinking on this subject may be far-reaching, concerning not only a true understanding of chest disease, but also the appropriate advice or treatment given to a patient, or even questions of medico-legal interest.

THYREOTOXIC GOITRE.

In thyreotoxic goitre, partial thyreoidectomy is often dramatically successful. In perhaps the majority of cases operation is the treatment of choice. Surgeons have had ample opportunity of observing the immediate effects, but very few observations appear to have been made of the later results of operation. Medical practitioners are perhaps too apt to regard the early post-operative condition as the permanent one. There are many features requiring prolonged investigation; for example, the progress of the cardiac condition and the state of the endocrine balance. It has been said, for instance, that partial thyreoidectomy does not cure thyreotoxicosis unless it is sufficiently radical to induce a state of subthyreoidism. To test the truth of this assertion alone would require many years' research. Harold H. Rosenblum and Samuel A. Levine have recently completed a series of observations on a group of 69 patients who had suffered from thyreotoxicosis and cardiac failure and had been treated by operation.[1] Their investigations extended over a period of some four and a half years; they were concerned almost solely with the progress of the cardiac disorder.

Two of the patients died shortly after operation; six others died after an average period of two and a half years, though they had all been restored to normal or a condition enabling them to resume a moderately active life. There was no pronounced alteration in the blood pressure after operation, nor was there any great change in the size of the heart. Rosenblum and Levine suggest that cardiac enlargement is due to associated cardiac disease and not directly to hyperthyreoidism.

There were 32 patients who had suffered from persistent auricular fibrillation before operation; 24 of these were adequately reexamined. Eleven of them had mitral stenosis and did not lose their abnormal cardiac rhythm spontaneously; seven others continued to suffer from auricular fibrillation; one was relieved by the use of quinidine, and five were spontaneously cured. Rosenblum and Levine remark that it is useless to give quinidine before operation in these cases and dangerous to give it after operation if the patient is suffering from mitral stenosis.

In five of six cases paroxysmal auricular flutter disappeared after operation; in the sixth it recurred in association with other evidence of hyperthyreoidism. There were nine patients suffering from anginal paroxysms; in every case the paroxysms either disappeared or became much milder and recurred at longer intervals. Rosenblum and Levine believe that all these patients were affected with disease of the coronary arteries and that the angina was the result of the added strain imposed on the cardiac mechanism by the greatly increased basal metabolic rate.

The relief from the symptoms of cardiac failure after operation was pronounced. In this regard it is of interest to note that the condition of a patient, suffering from cardiac failure without any apparent disturbance of the thyreoid gland, was strikingly improved by subtotal thyreoidectomy. Rosenblum and Levine suggest that the operation may be of value in various forms of cardiac disease.

In this series of cases investigations revealed that the improvement appearing as an immediate effect of operation "was extremely well maintained". It is important to note that the average age of the patients was 50·7 years at the time of operation. A higher incidence of obvious organic heart disease might reasonably be expected among patients of this age. Rosenblum and Levine stress the rarity of heart failure among young patients suffering from hyperthyreoidism, and point out that, when there is heart failure, there is nearly always evidence of organic heart disease. This important contribution serves to emphasize the fact that a vast amount of work remains to be done on the subject.

[1] *The American Journal of the Medical Sciences*, February, 1933.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Plasmodium Ovale.

S. P. JAMES, W. D. NICOL AND P. G. SHUTE (*Annals of Tropical Medicine and Parasitology*, July 14, 1932) record the results of their inquiry into the morphology and bionomics of *Plasmodium ovale* Stephens. Stephens first described this parasite in 1922. In 1927, in collaboration with Owen, he made further studies of the parasite. In 1930 the findings of Stephens and Owen were confirmed by Yorke and Owen. These latter observers showed that the morphological features of *Plasmodium ovale* in its asexual stages remained constant when it was passed from person to person by direct inoculation with infected blood. The organism studied by James, Nicol and Shute was obtained from a patient who had been infected by direct inoculation from the blood of a patient from the Belgian Congo. They were able to observe the phenomenon of exflagellation and to transmit the infection by means of an intermediate host, the anopheline mosquito. They also observed various developmental stages in the parasite's life history in the body of the mosquito. The fever induced in man by the *Plasmodium ovale* is tertian in periodicity. From their observations on the parasite in its sexual and asexual cycles, the authors conclude that it is a separate species of human plasmodium.

The Specificity of Antiviruses.

A. BESREDKA (*Journal of Immunology*, November, 1932) discusses the results of experimental investigations undertaken by other workers to decide whether antiviruses act as does ordinary broth by increasing the resistance of the cells with which they come in contact or by conferring on the cells a real local immunity varying according to the bacterium with which they were prepared. He discusses in the first place the effect produced by antiviruses *in vitro* on their respective bacteria. He states his view that the inhibitory action of antiviruses plays a secondary rôle in the process of local immunization, which essentially depends on the affinity of the antivirus for the respective cells of the tissues. The author states that the experimental work reviewed supports his contention that antiviruses are endowed with a specificity as strict as that of the bacteria from which they come.

Bovine Tubercle Bacilli in Sputum.

W. MELROSE CUMMING AND WINIFRED M. FOSTER (*Journal of Pathology and Bacteriology*, January, 1933) report the result of their investigations into the occurrence of the bovine type of tubercle bacilli in sputum. Material was from all patients with pulmonary tuberculosis admitted to institutions for tuberculous patients in Bradford; from patients with pulmonary tuberculosis in whom a definite history of a previous glandular lesion could be obtained; from children with pulmonary tuberculosis; from patients with pulmonary tuberculosis who were likely to be in contact with tuberculous cattle; and from patients whose pulmonary tuberculosis was complicated by lupus. Details of the methods of isolation and identification of the organisms are given. Modified Löwenstein's medium and Dorset's medium were in regular use. Strains were isolated from three hundred and ninety-one patients, and fourteen of these were proved to be of bovine type. The presence of a previous glandular lesion was the outstanding feature of the investigation. It was noted in all but five of the fourteen cases and is regarded as strongly suggestive of an alimentary route of infection. The authors sum up the result of their investigation as follows. From two hundred and eleven unselected cases of pulmonary tuberculosis in a northern urban industrial population six bovine strains were isolated; five of these had previously had a glandular lesion. Of fifty-one cases of pulmonary tuberculosis in the north of England (including the Bradford cases) in which there had been definite evidence of a previous glandular lesion, ten have been shown to be due to the bovine type of the bacillus. Of fourteen similar cases in the south, one has been shown to be due to the bovine type. Of fifty-nine cases of pulmonary tuberculosis in the north of England occurring in children under sixteen years of age, one has been shown to be due to the bovine type. Of eleven cases of pulmonary tuberculosis occurring in patients coming much in contact with cattle, two have been shown to be due to the bovine type. In any given case of pulmonary tuberculosis the presence of tubercle bacilli of bovine type seems to be of definitely unfavourable prognostic significance.

Immunity to Vaccinia Virus.

R. W. FAIRBROTHER (*Journal of Pathology and Bacteriology*, January, 1933) gives details of some observations on the immunity induced in rabbits by inoculating serum-virus mixtures by the intracerebral route. Animals surviving the inoculations were tested: (i) by determining the presence of antiviral bodies in the serum by the neutralization test, and (ii) by subsequent intradermal inoculation of the animals with an active virus. In the majority of animals a high degree of immunity resulted and could be demonstrated at least ninety days after the inoculation. In occasional animals of poor general condition the immunity was not so marked. In most instances no definite systemic reaction followed the inoculation, but when a comparatively large dose of virus was used, marked weakness of the hind legs with general tremor occurred eight to ten days after inoculation; but these animals made a complete recovery. It was noted that the mixtures of virus and serum which produced these reactions were inactive when introduced by the intradermal route. When the inoculation was made into the brain, a very small dose of virus was necessary to evoke a high degree of immunity. The relative merits of the subcutaneous and intracerebral routes were studied and it was noted that little, if any, immunity was produced by the former. This suggests that the production of immunity depends on some action of the living virus on the tissues. The results of further experiments strongly suggest that phagocytosis is an important defence mechanism of immune animals against vaccinia virus. It was found by intracerebral inoculation that the action of immune serum on an active virus depends on the time and temperature allowed for the contact *in vitro*. Also the action of the serum is more obvious when leucocytes are added. This was regarded as an indication that the serum sensitizes the virus and so renders it more susceptible to destruction by cells.

Susceptibility in Poliomyelitis.

CLAUS W. JUNGEBLUT (*Journal of Immunology*, February, 1933) has investigated the mechanism underlying susceptibility in infantile paralysis. Previous studies had suggested the influence of some genetic factor on the neutralizing power of human sera and led to an investigation as to whether a similar relationship between blood groups and neutralization could be detected. A number of adult human sera were also examined for virucidal power. The technique for the neutralization test was as follows: 0·6 cubic centimetre of undiluted, non-inactivated serum was mixed with 0·6 cubic centimetre of a 10% virus suspension. The mixture was incubated for one and a half hours at 37° C., kept over night in the ice-box, and the following morning injected intracerebrally in one cubic centimetre volumes into monkeys. The animals were observed daily for fever and paralytic symptoms over a period of at least four weeks. Each test included a monkey inoculated with the virus alone as a potency control. Virus suspensions were prepared from no less than six to eight pooled poliomyelitic cords, carefully selected from animals which had developed complete paralysis after a short incubation period. The author summarizes his results as follows. A series of thirty normal adult human sera collected from metropolitan individuals and consisting of ten O, ten A, and ten B sera, were tested for neutralizing power against poliomyelitis virus. The incidence of neutralization obtained with the different blood groups was: Group O, 60%; Group A, 30%; and Group B, 80%. The titre of three lots of pooled neutralizing O, A and B sera

was determined by quantitative titration. The highest dilution capable of completely inactivating the virus was with the O sera one in twenty, and with A sera one in five, while the B sera neutralized up to a one in sixty dilution. In view of the wide quantitative and qualitative variations in neutralizing power among normal adult human sera, depending, as it seems, in a large measure on the blood group of the individual, the virucidal test must be considered basically unsound as a means of determining previous exposure to the virus in epidemiological studies.

A Method for Securing Clear Sera from the Milk of Cows and Goats for Agglutination Tests.

IVAN C. HALL AND ROBERT LEARMONTH (*Journal of Infectious Diseases*, January-February, 1933), finding that many owners of cows and goats objected to having their animals bled when the necessity arose to test these animals by the Zammit test, investigated the possibility of removing fats from milk so that clear sera could be obtained from this source. Ether and benzene were found to be undesirable for the purpose, owing to their low specific gravity and high volatility, but carbon tetrachloride and chloroform were found satisfactory. In practice, test tubes for the collection of milk samples are corked in the laboratory, each containing a few cubic centimetres of either solvent and a small amount of rennet extract. Milk from the animal is collected directly into the tube. The tubes are then placed in the incubator at 37° C. for about an hour to coagulate the casein. They are then centrifuged for fifteen minutes at two thousand revolutions per minute to separate the milk serum, which will be found in a perfectly clear deep layer at the top, with a compact curd in the middle and the solvent at the bottom. Serum collected in this way may be preserved indefinitely owing to the germicidal action of the solvents, but the stability of agglutinins has not been tested.

HYGIENE.

Eye Colour and Disease of the Upper Respiratory Tract.

WILLIAM M. GAFAFER (*American Journal of Hygiene*, November, 1932), in a research on the common cold, has observed 343 adults of both sexes for thirty-five weeks, from September, 1929, to May, 1931. All attacks of upper respiratory disease (common cold) were recorded. No significant difference showed between the group with brown eyes (175 in number) and the group with blue eyes (168 in number), whether as to frequency, severity or type of attack of upper respiratory disease. Of the attacks 438 occurred in the brown eyed, 463 in the blue eyed; 21% of the former and 31% of the latter had more than three attacks. Of the attacks in the brown eyed 11·4% required treatment in bed, while 15·1% of the blue eyed were similarly marked. On the other hand, 60·2% of the attacks in the brown eyed lasted ten days or more, while 59% of the attacks in the blue eyed reached this duration.

Pulmonary Tuberculosis Complicating Glandular Tuberculosis.

W. MELROSE CUMMING (*Tubercle*, March, 1933) contests the prevailing belief that pulmonary tuberculosis rarely follows a gross tuberculous glandular lesion, and that when it does, the pulmonary lesion tends to run a benign course. He has investigated fifty-eight patients with pulmonary tuberculosis, whose history revealed previous gross glandular lesions; of these, nine were due to the bovine type of bacillus. In no less than twenty the glandular lesion appeared only in adult life. Thirty-two patients had been operated on for the removal of the affected glands, of whom twenty-five at the time of investigation had a bad prognosis from the point of view of the pulmonary lesion. Of the twenty-six who had not been subjected to operation, a bad prognosis was offered in fifteen, suggesting that operation was not a factor in preventing a later pulmonary lesion, nor did it determine a more benign type. In forty-one patients the lung lesion was of the usual apical type, and in only two was the prognosis good. In twelve patients the lesion was of the root-spread type, and in four the prognosis was good. This suggests that the type of lung lesion has a bearing on prognosis. Among the thirty-eight patients who had a glandular lesion in childhood, in only eight was the bovine tubercle bacillus isolated from the sputum. If the organism in the sputum is that which caused the earlier glandular lesion, then the proportion of bovine strains found in this series is far below that generally found in glandular tuberculosis in childhood. To explain this discrepancy the author makes the suggestion that the higher rate of human type infections here shown is the result of a fresh exogenous air-borne infection, by a strain of the tubercle bacillus other than that which caused the original glandular lesion.

The Value of Löwenstein's Blood Culture in Experimental Guinea-Pig Tuberculosis.

P. MANTEUFEL AND E. KOTTMANN (*Münchener Medizinische Wochenschrift*, January 27, 1933) conclude that the value of cultures for the diagnosis of tuberculosis in living guinea-pigs is limited, and that at present there appears to be very little hope of shortening the time for obtaining conclusive proof by diagnostic animal research. The same result was also arrived at in rabbits infected with tuberculosis. Negative results were obtained in guinea-pigs with the *Bacille Calmette-Guérin* infection, 65 cardiac punctures and 250 cultures being made. From this research it appears that the tubercle bacillus rarely reaches the circulation in this affection, although it cannot be totally excluded. *Post mortem* 50% of these cases gave positive findings, and probably more would have been obtainable for culture. The bacteriæmia in guinea-pigs *post mortem* was almost as constant a finding as in the rabbit. The authors conclude that there appears to be no reason for the assumption that the many positive blood cultures obtained in the laboratories of Löwenstein have been mistaken for non-pathogenic acid-fast bacilli.

Bath Deaths.

TANNHÄUSER (*Münchener Medizinische Wochenschrift* November 18, 1932) quotes a case of a healthy man who noticed urticarial patches and wheals on face, hands and feet after washing himself with cold water. One day, after bathing in a cold lake, the above symptoms recurred, with pain in the stomach and an urge to defæcation, during which he collapsed and remained unconscious for half an hour. The pulse was very slow and thready. After one hour he felt quite well again, but a good coloration returned only one and a half hours later. This case of cold urticaria with the associated shock is possibly due to failure of the temperature regulation. The shock might be caused by histamine absorption from the urticarial wheals. It is therefore easy to understand how drowning may occur in cases of the type described.

Pneumococci and Specific Pneumonia Therapy.

PAUL MARTINI (*Münchener Medizinische Wochenschrift*, January 20, 1933) finds that the pneumococcus of Fraenkel is the predominating causative organism of pneumonia. Till now 32 different types of pneumococcus have been described, Types 1, 2 and 3 being constant and much more virulent than Types 4 to 32. Types 1 to 3 are practically found only in cases of pneumonia, whereas the remaining types are very often found in healthy people. Pneumonia is either the result of an infection with a virulent type of organism or is due to lowered body resistance, and here a pneumococcus of a less virulent type can cause pneumonia. Fluctuating powers of virulence of the pneumococci do not occur. Even today we do not know why one person becomes ill with pneumonia and the other person escapes, even if exposed to the same conditions. Probably here constitutional as well as immunization and allergic factors play an important rôle. Typical immune bodies are formed which drive the pneumococcus from the blood stream; similar results are obtained with a corresponding specific serum. Administration of the specific type of serum hastens the crisis and prevents further spread of the lung process, but it does not cause retrogression. The earlier the specific serum is used, the better are the results. Polyvalent anti-pneumococcal sera are unsatisfactory.

Special Articles on Treatment.

(Contributed by Request.)

X.

THE TREATMENT OF SCARLET FEVER.

FORTUNATELY, acute toxic and septic cases of scarlet fever are much less common than they were thirty years ago. Death in the first few days of scarlet fever is now comparatively rare. In spite of this, scarlet fever is a more difficult disease to manage; for the sequelæ are more common and very much more troublesome. The prognosis of scarlet fever as regards survival is, as a rule, excellent, but even after the mildest case the patient may be left with damaged kidneys and chronic ear discharge. The management of convalescence, always important, is now more important than ever. The aims in treatment may be summarized under three heads, namely: (i) lessening the toxæmia; (ii) prevention, if possible, of the septic element; (iii) treatment of complications.

Lessening the Toxæmia.

We now have a powerful agent in helping us to combat toxæmia, namely, a specific antitoxic serum. Scarlet fever antiserum has been in use at the Infectious Diseases Hospital, Fairfield, for the last seven years. We have come to one definite conclusion. It should be used in the early stages of the disease in every severe case.

The beneficial effects of scarlet fever antiserum are most strikingly seen in the early toxic cases with high fever, brilliant rash, and red, injected throat. The effect of an adequate dose (20 or 30 cubic centimetres) of a specific antiserum is dramatic. The temperature falls in twenty-four to thirty-six hours, the rash fades quickly, and the patient looks and feels more comfortable. When the disease has progressed for four or five days, and the septic element has developed, and rhinorrhœa, swelling of the fauces, and enlargement of the cervical glands are present, the results are not nearly so satisfactory. I consider, nevertheless, that it is worth giving in these cases. There is some evidence against giving the serum in mild and moderately severe cases. It is held that the complications of scarlet fever are due to fresh infections with other streptococci, or, in some cases, to a reinfection with the original streptococci. Now the passive immunity induced by giving serum is, in most cases, a temporary one, whereas the active immunity produced by the patient's own antibodies is more permanent.

Theoretically, in mild and moderately severe cases, patients who have been given serum should be more likely to develop troublesome sequelæ, especially if they are kept in an acute ward, where they are liable to become reinfected from time to time. Practical experience seems to bear this out. The routine adopted at Fairfield is to give serum to all children who have a temperature of 38·9° C. (102° F.) or over, or who look at all sick. The usual dose is 20 cubic centimetres, given intramuscularly. Occasionally it has to be repeated. Adults as a rule do quite well without serum; but in severe cases they are made more comfortable.

Another method of combating toxæmia is by hydrotherapy. Free fluids by the mouth with the efficient use of sponging can produce very good results in cases of ordinary severity. If the soreness of the throat prevents the child from receiving the required amount of fluid, the administration of saline solution by the rectum should be tried. Between 1,100 and 1,400 cubic centimetres (40 and 50 fluid ounces) of fluids in 24 hours should be given as a minimum to a sick child.

External hydrotherapy stimulates the free circulation in the skin, thus lessening toxæmia as well as adding to the patient's comfort.

Prevention of the Septic Element.

Theoretically, by neutralizing toxin and preventing local damage, an efficient dose of antiserum given early in the disease is the surest way of preventing ingress of other streptococci. Practically this is found to be so. Therefore, serum-treated patients will, as a rule, not develop the signs of acute local sepsis, at any rate not in the early stages of the disease. Later on, of course, when the immunity has probably disappeared, this may be so.

One cannot stress too much the importance of efficient cleansing of the throat and mouth. Apart from its soothing effect, the saline douche is the most valuable method of cleansing the throat. This douching, combined with swabbing the gums and teeth with "Listerine", is the routine method at Fairfield. It is done four-hourly; but if the severity of the throat affection warrants it, two-hourly douches are administered.

The Treatment of Complications.

Adenitis of Convalescence.—Some swelling of the glands of the neck occurs at various times in most cases of scarlet fever, and is treated on ordinary lines. If abscess formation threatens, never be in a hurry to open the swelling. Early incision may have bad results, such as extensive sloughing and ulceration of the skin, which otherwise may have been avoided. If sloughing is going to occur in any event, an incision will not prevent it, and may hasten it. Wait until you are sure pus is present, then wait for another twenty-four hours.

Otitis Media.—The development of earache accompanied by a rise in temperature, warrants immediate examination of the drum. If the drum is bulging, do not wait, open under general anæsthesia. If the drum is not bulging, but remains red and inflamed for twenty-four hours, open at the end of that time. Once the ear is discharging, an efficient ear toilet may prevent the formation of a mastoid abscess, and will certainly lessen the duration of the discharge. If the signs become suggestive of a mastoid abscess, the problem is when to operate. I think that there is an optimum time in these cases. If signs of a mastoid abscess develop very soon after the ear has commenced to discharge, and there is a high temperature and copious blood-stained serous discharge from the ear, I fancy that the results are better if the operation is left for twenty-four hours. The question, however, is one for the specialist.

Nephritis.—For the year ending July, 1932, there were 77 cases of nephritis following scarlet fever, or 4·1%, at the Hospital for Infectious Diseases, Fairfield. In 1922 there were four cases of nephritis, or about 0·25%. This gives some idea of the increasing severity of scarlet fever in the later stages of the disease. Fortunately, the immediate prognosis of scarlatinal nephritis is usually good; I am inclined to think also that, in children anyway, the after-effects have been exaggerated. A large percentage undoubtedly clear up completely.

The treatment of scarlatinal nephritis is straightforward. Many of the measures in the routine treatment of scarlet fever are carried out largely with a view to the prevention of nephritis. Thus we have: (i) prolonged rest in bed, even in mild cases; (ii) careful arrangement of the diet; (iii) stimulation of other excretory organs, the skin and bowel; (iv) administration of fairly large amounts of fluid to dilute toxins as they pass through the kidneys.

The urine should be examined every second or third day in scarlet fever, and at the first sign of nephritis treatment should be commenced. This will consist mainly in making more strict the preventive measures mentioned above. Thus the patient will be kept in bed for a longer period, usually about two months. When there are a few red blood cells only, a trace of albumin and a few casts in the urine, the patient may be allowed out of bed. Increasing exercise and fuller diet will be gradually introduced; the urinary findings will show how much can be done in this direction without harm.

The diet in the early stages of nephritis should be reduced to the simplest form. Salt is excluded. A saline purge should be given at least once a day and, if necessary, twice a day. Hot packs and hot air baths may be used. If there is much œdema, the amount of fluids may be reduced; but in my opinion drastic reduction of fluids is harmful. Diuretics also are unnecessary and may be harmful.

When the tonsils are grossly septic, they may be removed at a comparatively early stage of nephritis. We have removed them at Fairfield as early as the third week after the onset of nephritis. The results are undoubtedly good and the operation in suitable cases hastens the clearing of the urine.

The results of kidney function tests are almost invariably satisfactory in scarlatinal nephritis; but a low urea concentration is occasionally seen in adults; in these cases I think the ultimate prognosis is not good.

While on the subject of nephritis, it is worth while considering the use of a saline purge instead of calomel in the first days of scarlet fever. It has been clearly proved that calomel has a toxic action on the kidney, and therefore I am of the opinion that it should not be given.

Scarlatinal Rheumatism.—In adults scarlatinal rheumatism is a particularly worrying and distressing complication. In some it will persist as vague muscle and joint discomfort all through their stay in bed. There may be no joint swelling; but I am of opinion that these patients should have a slow convalescence and should be kept in bed until all signs of rheumatism have subsided. Even then some weeks elapse before they are fit to return to any strenuous work. I have found this to be so, particularly with nurses.

Sodium salicylate eventually causes subsidence of the joint pains; but for the immediate relief of pain, there is nothing so good as aspirin.

Relapsing Cases.

I have purposely left to the end the discussion of a type of case which is unfortunately becoming more common. In these no definite complications occur, but the child never looks quite well. There may be slight swelling of the glands in the neck, slight persistent rhinorrhœa, and always some reddening and inflammation of the tonsils and fauces. These patients are liable to relapses and second rashes, even as late as the fourth and fifth week of the disease. They also remain infectious for a considerable period. Removal of tonsils and post-nasal growths, or washing out of infected antra will improve them. Tonsillectomy, however, should be done with care. Marked secondary anæmia is common, and, in some cases, there is a possibility of endocarditis. Vitamin therapy may be useful in these cases.

Summary.

The general lines of treatment in an ordinary case of scarlet fever may be summarized as follows:

1. Give a saline purge and commence routine treatment of the throat and mouth.

2. Give antitoxin if necessary.

3. Give a free fluid diet for the first forty-eight hours, then semi-solids until the throat is clear; later, give green vegetables, eggs and fish, and plenty of fruit. At the end of the third week, if there is no albumin, red meat can be added.

4. Children should be kept in bed the full three weeks; but adults may be allowed up about the seventeenth or eighteenth day, if there is no albuminuria.

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Acting Medical Superintendent, The Hospital for Infectious Diseases, Fairfield, Victoria.

British Medical Association News.

THE KATHERINE BISHOP HARMAN PRIZE.

THE Council of the British Medical Association is prepared to consider an award of the Katherine Bishop Harman Prize in the year 1934. The value of the prize is £75.

The purpose of the prize, founded in 1926, is the encouragement of study and research directed to the diminution and avoidance of the risks to health and life that are apt to arise in pregnancy and child-bearing. Competitors are left free to select the work they wish to present, provided this falls within the scope of the prize.

Any medical practitioner registered in the British Empire is eligible to compete.

Should the Council of the Association decide that no essay submitted is of sufficient merit, the prize will not be awarded in 1934, but will be offered again in the year next following this decision, and in this event the money value of the prize on the occasion in question shall be such proportion of the accumulated income as the Council shall determine. The decision of the Council will be final.

Each essay must be typewritten or printed in the English language, must be distinguished by a motto, and must be accompanied by a sealed envelope marked with the same motto and enclosing the candidate's name and address.

Essays must be forwarded so as to reach the Medical Secretary, British Medical Association House, Tavistock Square, London, W.C.1, not later than December 31, 1933.

Inquiries relative to the prize should be addressed to the Medical Secretary, Dr. G. C. Anderson, at the Medical Department, British Medical Association House, Tavistock Square, London, W.C.1.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Reynolds, Preston Guy, M.B., B.S., 1931 (Univ. Melbourne), Hospital for the Insane, Beechworth.

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association announces that the following books have been added to the library of the Branch:

From the "Hastings Collection", part of the library of the Worcestershire Medical Society, and containing many volumes presented by the late Sir Charles Hastings, founder of the Association (donated by the Parent Association): "Opera Omnia Medico-Practica et Anatomica", Georgii Baglivi, 1714; "Elementa Chemiae", Tomus Primus et Secundus, Hermannus Boerhaave, 1732; "Sepulchretum sive Anatomia Practica", Tomus Primus et Secundus, Theophili Boneti, 1679; "Illustrations of Elementary Forms of Disease; Pathological Anatomy", Robert Carswell, 1838; "Opera Omnia Medica" (Londini), dedicated to Queen Caroline, Johannis Freind, 1733; "Phthisiologica seu Exercitationes de Phthisi" (Londini), Richardo Morton, 1689.

"Roentgen Interpretation", Fourth Edition, 1931, G. W. Holmes and H. E. Ruggles; "Recent Advances in Radiology" (illustrated), 1931, Peter Kerley; "Homogeneous X Radiation and Living Tissues", Warnford Moppett; Clinical and Pathological Papers, Mental Hospitals Department, New South Wales, Volume IV, Parts 1 and 2, 1932; "The Present Position of Deep X-Ray Therapy", A. T. Nisbet; "Sanitary Law in Question and Answer", Porter and Fenton; "Egypt, the Home of the Occult Sciences", T. G. Garry; "Materia Medica", Hale-White (Douthwaite); "Heart Disease", C. Bramwell; "The Critical Age of Woman", W. A. Gallichan; "Cæsarean Section", Ivens-Knowles; "Urology in Women", E. C. Lewis; "The Diabetic A B C", R. D. Lawrence; "Clinical Notes on Disorders of Childhood", D. W. Winnicott; "The Mind in Action", A. C. Garnett; "Towards National Health", J. A. Delmege; "Life of Mendel", H. Iltis; "Handbook of Bacteriology", J. W. Bigger; "Diseases of the Kidney", Ball and Evans; "Hypertension and Nephritis", A. M. Fishberg.

The Royal Australasian College of Surgeons.

SIXTH ANNUAL GENERAL MEETING.

THE sixth annual general meeting of the Royal Australasian College of Surgeons was held in Sydney from April 10 to April 13, 1933. It was attended by, approximately, two hundred Fellows of the College, including representatives of all the Australian States and of the Dominion of New Zealand.

Proceedings began at 8.15 p.m. in the hall of the Conservatorium of Music, when His Excellency the Governor-General of Australia declared the meeting open. The ceremony of formal admission of new Fellows followed His Excellency's address, after which Professor F. Gordon Bell, Professor of Surgery in the University of Otago, delivered the second George Adlington Syme Oration on "Hospital Problems and the Education of the Surgeon". This address will be published in the next issue of *The Australian and New Zealand Journal of Surgery*. Invitations for this meeting were issued to all members of the British Medical Association residing in or near Sydney, and also to members of the hospital boards and others interested in the subject of the address.

On Tuesday, Wednesday and Thursday clinical and operative demonstrations were given at the various Sydney hospitals, which were much appreciated by all visiting Fellows.

Standard of Requirements for Fellowship of the College.

At the annual business meeting, held on April 11, Fellows present unanimously approved of a special resolution submitted by the Council of the College to alter the Articles of Association in such a manner that a radical change in the requirements for fellowship could be introduced. The reasons for the proposed amendments of the Articles of Association and the consequential alterations in the regulations of the College which would follow their adoption were explained by the Honorary Secretary (Dr. Alan Newton). A synopsis of his address is as follows:

Perhaps the chief object of this College is to foster in Australasia higher efficiency in surgical work and, in the words of Sir Louis Barnett at the Brisbane Congress in 1920, "to bestow a hall-mark on the efficient". There seems little doubt that, if this hall-mark is bestowed only upon those who are unquestionably worthy, the general standard of surgical capacity will be raised, as more and more men would undergo the training necessary to qualify them for the hall-mark bestowed by fellowship of this College.

The founders of the College recognized that, if the general level of surgical efficiency in Australasia was to be raised, it was necessary for the College to formulate a minimum standard of surgical training. The Council, therefore, in 1927 adopted the two following principles: (*a*) The minimum period of post-graduate practical training in surgery should be five years. (*b*) A senior surgical qualification must be possessed by all candidates applying for fellowship after the lapse of a period of five years from the date of foundation of the College.

In other words, the College, since its foundation, has maintained that an adequately trained surgeon must have a thorough knowledge of the basic principles of surgery, as shown by the possession of a senior surgical qualification, and, what is just as important, he must have served a five years' period of apprenticeship in the practical application of these principles.

It was, of course, recognized that there must be some means of testing the suitability of a candidate for election to fellowship, and, at its foundation in 1927, the College adopted a system based upon that of the American College of Surgeons. Under this system, a candidate must be nominated by three Fellows and must give the names of five other medical practitioners, three of whom must be surgeons, as references. The five referees are then asked to fill in a confidential report concerning the surgical judgement and ability of the candidate and his moral and ethical standing. The application, together with the reports of the referees, is then considered by the State or Dominion censors, who report to the Council of the College, which then decides whether to admit, reject or defer the application, pending the submission of a series of case histories.

After six years' experience of this system the Council has come to the conclusion that it is now unworkable. It has found that it is impossible to insure that a similar standard is maintained in the various Australian States and in the Dominion. It has found that it is impossible adequately to assess the professional capacity of a candidate by the perusal of the written reports of referees. It is informed by the Censor-in-Chief that it is not possible for him to determine the merits of a candidate by examination of a series of case histories which he has submitted. It recognizes that the system of nomination occasionally places its Fellows in an invidious position. The Council fears that, if this system be continued, not only may there be undeserved admissions, but also there may be unjust rejections. After prolonged consideration of the whole problem during the past two years, the Council has formed the opinion that an essential feature of a system of admission to fellowship of this College must be a personal interview with a board of censors. In order to insure that there is a uniform standard of admission, the Council proposes that there should be one board of censors for the whole of Australia and another board of censors for the Dominion of New Zealand. These boards shall meet at such times and places as the Council may direct. Each board shall be empowered to submit each candidate to tests of his knowledge and ability, but it is proposed that, in general, the interview with the board of censors shall be similar to the last examination for the diploma of membership of the Royal College of Physicians, that is, the censors and the candidate would sit round a table, and that the censors should ask the candidate such questions as are considered necessary. It seems probable that, in this way, such a board could satisfy itself as to the suitability of the candidate for election as a Fellow, in so far as the degree by which he has profited from his practical training is concerned.

This proposal has been submitted to the various State Committees and the Dominion Committee, and has received general approval.

Consequent upon the adoption of the alterations in the Articles of Association, the future requirements of a candidate for fellowship of the College will be as follows:

(*A*) A five-year period of post-graduate training, which, broadly stated, should consist in:

1. At least one year's training as a resident medical officer in an approved hospital.

2. Obtaining a senior surgical qualification, which must entail a primary examination in anatomy and physiology and a final examination in surgery, surgical anatomy and surgical pathology. The primary and the final examination need not necessarily be passed in the same university or college of surgeons.

This regulation will not be enforced: (*a*) In the case of candidates who have obtained a senior surgical qualification, approved by the Council, prior to February 5, 1932. Such candidates will be considered qualified, in this respect, for fellowship of the College, despite the fact that the senior surgical qualification does not entail a primary examination in anatomy and physiology. (*b*) In the case of candidates for fellowship in ophthalmology or laryngo-otology, such candidates must possess a senior surgical degree or diploma in ophthalmology or laryngo-otology approved by the Council. Candidates for fellowship of the College who take advantage of this regulation, must describe themselves as fellows in ophthalmology or laryngo-otology, as the case may be, for example, F.R.A.C.S. (Oph.) or F.R.A.C.S. (L.-O.). It has been represented to the Council that New Zealand graduates not only pass an examination in anatomy and physiology at the end of the third year of the medical course, but also must pass another examination in anatomy and physiology at the final qualifying examination in medicine. The Council has decided that this examination shall be con-

sidered a primary examination in anatomy and physiology under the College regulations.

3. A period of surgical apprenticeship spent in assisting a more senior surgeon in operative and clinical work. It is not necessary that this period of apprenticeship should be undergone exclusively in metropolitan hospitals. Credit will be given for work in university departments, in research, and in surgical work in other countries.

(*B*) Candidates who have undergone this training must apply, on an approved form, to the Censor-in-Chief for admission to an interview with a board of censors. The Censor-in-Chief, if necessary, after consultation with the board of censors, shall determine whether the training undergone by the candidate is adequate. If, in his opinion, the training is not adequate, he will indicate to the candidate the further work which is considered necessary in his case. Candidates deferred by the Censor-in-Chief can appeal to the Council against his decision.

(*C*) Candidates approved by the Censor-in-Chief will appear before the board of censors, which is empowered to submit them to tests of their knowledge and to require them to produce reports of the practical work which they have done.

(*D*) Candidates approved by the board of censors must then apply to the Council for election on a special form. Power is retained by the Council to make such inquiries as it thinks fit as to the ethical standing of each candidate before admitting him to fellowship.

The Council recognizes that it must shoulder the responsibility of creating an Australasian standard of surgical training, and preferred to set this standard in advance of that exacted elsewhere. It firmly believes that an improvement in the general level of surgical efficiency in Australasia can best be achieved by setting a high ideal of surgical training before the future surgeons of this country, but, at the same time, it fully appreciates the fact that the standard set must be attainable by those possessing normal ability and industry. It feels that the standard of admission, just outlined, fulfills both requirements. It has, naturally, been careful to examine the standard adopted in the light of all possible objections, some of which are summarized as follows:

1. It has been suggested that the senior surgical qualification regulation will close the door of fellowship of the College to all but a handful of applicants.

During the last ten years, approximately, one hundred and twenty Australasian graduates have obtained a senior surgical qualification of the type demanded. It is interesting to note that the practice of sitting for a degree or diploma of this type varies in the different States, as is shown by the fact that, subsequent to the foundation of the College, 37% of Victorian candidates, 31% of New Zealand candidates, and 31% of South Australian candidates possessed a senior surgical qualification of the type required, whereas 14% of New South Wales candidates and 8% of Queensland candidates were similarly qualified. The establishment of an M.S. degree in the University of Sydney and the standard set by the College will, doubtless, greatly increase the number of those in New South Wales who qualify themselves in this respect. These figures make it clear that the College doors will not be closed "to all but a handful" by the introduction of this regulation.

2. It has been stated that the time in acquiring an M.S. degree is unduly long. Investigation of the time spent by twenty candidates who have obtained the Master of Surgery degree at the University of Melbourne during the last five years reveals the fact that this degree is usually obtained in either three or four years after graduation. The exceptions are mainly in the case of candidates who have gone into general practice for some years and then have returned to the University in order to obtain a senior surgical qualification.

3. It is said that few will be able to afford to spend five years in surgical training. It has already been shown that one hundred and twenty men in the last ten years have undergone a training approximating in time to that demanded, but the Council hopes to make it easier for many more to undergo the necessary training. It proposes to use part of its surplus income in assisting men of ability to undertake this task. It wishes Fellows to realize that when paying their subscriptions, they are helping adequately to train their successors. This, by the way, was the chief reason which induced it to appeal for Fellows to contribute to the building fund, as a generous response would free the College from debt and enable it to begin this plan. The Council also proposes to use a large part of the Craig endowment, when it becomes available, in this manner.

4. It has been suggested that it is presumptuous on the part of this College to impose a further test upon candidates who have already obtained a senior surgical qualification. It is well known that the acquisition of such a qualification does not necessarily imply ability in practical surgery. The Council lays great stress upon the importance of a superimposed apprenticeship and feels that it is essential to have some test of the degree whereby the candidate has profited from it. It wishes to be sure that its "hall-mark" is reserved for vessels of silver and is not stamped upon those of pewter.

The Council has given special attention to the means whereby a candidate can undergo the surgical apprenticeship which it regards as so important in the training of a surgeon. It proposes to ask all clinical schools to make available positions of this nature, of which the maximum tenure must be not more than two or three years. The Melbourne Hospital has already created positions of this type. The whole energies of the College will now be devoted to this task. It is believed that positions will be made available, not only in clinical schools, but also in other metropolitan and in large country hospitals, which will enable a large number of men to undergo this apprenticeship.

The Council realizes that it is, unhappily, necessary to educate some members of the medical profession that the practice of surgery requires adequate and special training. It appeals to all Fellows to assist it in this task, and is gratified that, as a first step in this direction, unanimous approval has been given to the standard of surgical training.

Surgery of the Sympathetic Nervous System.

A preliminary report on the result of the investigations of the Victorian Committee appointed to investigate surgery of the sympathetic nervous system was presented to the meeting. It is expected that the final report will be completed at an early date. It will be published in a future issue of THE MEDICAL JOURNAL OF AUSTRALIA.

Report of the Hydatid Registry.

The Registrar, Roland A. H. Fulton, of Dunedin, reported that the total number of hydatid infections registered to date is 174, of which New Zealand has contributed 97 and Australia 77. His aim is to obtain the histories of one thousand cases, all with a complete account of the end results. It is hoped that all medical practitioners in Australia will assist this work by forwarding records of their cases of hydatid disease. Appropriate forms will be forwarded on application to the Head Office of the College, 6, Collins Street, Melbourne, or to any of the State Secretaries.

Admission of New Fellows.

At a meeting of the Council of the Royal Australasian College of Surgeons held in Sydney on April 10, 1933, the following candidates were admitted as Fellows:

(*a*) *Under the Old Regulations*:

New South Wales: Thomas Dixon Hughes, James Adrian Lawson, John Charles Ross, Roy William Whiston Walsh.
New Zealand: Arthur Gruchy Clark.
Queensland: Aubrey David Dick Pye, Reginald George Quinn.
Victoria: John Joseph Kelly, Jack Charles Lewis.

(*b*) *Under the New Regulations*:

New Zealand: Cyril Arnold King.
South Australia: Alan Harding Lendon.
Tasmania: George Albert Walpole.
Western Australia: Richard William Johnson, Hector Hamilton Stewart.

APPOINTMENT OF CENSOR-IN-CHIEF.

Owing to the death of R. Hamilton Russell, the President of the Royal Australasian College of Surgeons has given his approval to a recommendation made by the Executive Committee that Alan Newton act as Censor-in-Chief until the next meeting of the Council, that the vacancy thus created in the position of Honorary Secretary and Treasurer shall be filled by Julian Smith, Junior, and that G. R. A. Syme shall act as Honorary Assistant Secretary.

Correspondence.

THE CLOSED DOOR.

SIR: "High Priest", in a letter published in your journal of March 25, under the title "The Closed Door", protests against that regulation of the Royal Australasian College of Surgeons which provides that candidates for its fellowship must possess a senior surgical qualification.

The policy of the College concerning the future admission of Fellows is described in a report of its sixth annual general meeting, which is published elsewhere in this issue.

I wish to direct the attention of your correspondent to this report in the hope that, after reading it, he will appreciate the fallacies in his arguments and, in future, will serve, as high priest, at the high altar of surgical efficiency.

Yours, etc.,

ALAN NEWTON.

6, Collins Street,
Melbourne,
April 26, 1933.

EXPERIENCES IN RADIATION IN CANCER IN THE FEMALE PELVIS.

SIR: The paper by Dr. D'Arcy and Dr. Keatinge on this subject in the journal of May 6, 1933, contains much of interest. I am instructed by their results with patients suffering from cancer of the ovary. In work done prior to 1927 we noticed that this group could be treated with encouragement.

My impressions on the subject were published in THE MEDICAL JOURNAL OF AUSTRALIA of May 10, 1930, and in referring to the radiotherapy of the malignant ovary I suggested partial resection followed by deep therapy as a favourable course of treatment.

This note is prompted by the comment that the literature contains no reference to the matter. It is likely that other radiologists have reported similar experiences and a search in earlier papers might be rewarded.

Yours, etc.,

F. J. GWYNNE.

Auckland,
New Zealand,
May 15, 1933.

COAL MINER'S LUNG.

SIR: Dr. J. G. Edwards's criticism, in the journal of May 20, of opinions on radiography of coal miners' lungs flies wide of the mark. In his remarks about the radiological report on Case XII he has been misled by a misprint in the picture of the skiagram which is correctly labelled Figure III, and incorrectly Case XII and not Case XXII. Had he read the text he need not have continued in this error. His remarks about the need to employ an expert radiologist to interpret skiagrams such as those taken at Waterfall are equally unfortunate. Dr. Moore, who conducted this examination with me, is an experienced radiologist, and Dr. W. A. Edwards was good enough to read for us the majority of the skiagrams which were not normal, and it was noted with interest that his interpretations were very close to ours. But of these interpretations Dr. Moore and I wrote: "It is assumed that certain opacities seen in the radiographs of these cases have been caused by infective processes, frequently but by no means always tuberculous".

It is of great interest to read that there has been, in Sydney Hospital, extensive *post mortem* checking of the radiographic findings in pulmonary fibrosis. Does Dr. Edwards really refer to pulmonary fibrosis due to dust? If so, then it would be of no little help to his art if these cryptic post mortems were published. Will he excuse me if I am somewhat sceptical? When he goes on to infer that there has been much pathological work done at Broken Hill then I would quote the report of Dr. George for the year 1928-1929:

> Opportunities for studying the pathology of pneumokoniosis at Broken Hill and for correlating radiographic appearances with the actual *post mortem* condition of the lungs have been, and still are, extremely few. The Act does not provide for the *post mortem* examination of deceased beneficiaries. Post mortems are conducted only when a claim is made by the relatives of a deceased mine-worker that his death was due to dust disease. In these cases, even if a compensable condition is found present, there is no radiograph taken during life to compare with the *post mortem* findings.

I have yet to see a report on the pathology of the lungs of Broken Hill miners; there is certainly no *post mortem* evidence that the correctness of intrepretation was found to be high in the nineties. It seems probable that many of the men diagnosed by the radiographer as silicosis and tuberculosis did not die of tuberculosis.

Dr. Edwards says that "the X ray picture is one of projected pathology; it takes many years and the study of many thousands of cases before proficiency is attained in its interpretation", and judging by the gross radiographic errors I have seen in my limited series of lungs, errors made by men in whom Dr. Edwards has every confidence, I can only agree and hope that our radiographers will live long enough to acquire merit from complete understanding. Meanwhile, my naïve idea is that there is a better way to knowledge that will replace the deductive reasoning of the radiographers and their almost metaphysical training that results (but, alas, in so few) in the development of a flair for the correct detection of pulmonary dust fibrosis and tuberculosis; it is that we should correlate our radiographs with the lungs and to understand that we cannot jump blindly from one industrial condition to another.

I have objected for many years to the labelling and penalizing of workmen as tuberculous when there is no clinical reason for this, and have found that the belief of the radiographer that he could separate simple tuberculosis from silicosis is untrustworthy. In Australia the practice of radiography has put a blight on the study of dust diseases. I found the South African "rough justice", which is all they boast of, a better thing than our radiographic despotism untempered by post mortems. But these contentions only stress the need for a collection of lungs affected by industrial conditions and their intensive study. I beseech all those interested, if necessary, to forgive me my views (they may change) and to aid me in this matter, urging that only one interested will deal effectively with this difficult material.

Yours, etc.,

CHARLES BADHAM,
Medical Officer of Industrial Hygiene.

Sydney,
May 24, 1933.

PLACENTAL INFECTION FOLLOWING THE INDUCTION OF LABOUR.

SIR: In your issue of the 22nd ultimo, Dr. Frank A. Nyulasy asks for further information relative to the causation of convulsions by *Bacillus coli*. I agree with him that it is remarkable that this subject has escaped

the attention of the bacteriologists almost entirely, but on page 582 of the 1929 edition of Thursfield and Patterson's "Diseases of Children", these authors, writing of pyelitis, stated: "The onset is usually sudden with high fever and within a few hours the infant gives the impression of being seriously ill. Convulsions and vomiting and even rigors may occur at this stage." Over 90% of these cases of pyelitis show coliform infection of the urine. Again, on page 652, the same authors state: "Most important of the causes of symptomatic convulsions in young children are the toxins produced in the body in cases of infection by the *Bacillus coli* and other organisms, etc." It seems doubtful whether *Bacillus coli* infections can produce convulsions in adults. They must be highly exceptional if they occur at all in coli infections of the urinary or biliary tracts, but there seems to be no doubt in the minds of the clinicians that they can produce convulsions in children. Owing to the infection of the placenta and the fœtus with *Bacillus coli* in cases of eclampsia, it seems advisable to exclude infections of the fœtus by *Bacillus coli* before attributing convulsions of the new-born to the toxæmia of eclampsia.

Yours, etc.,

WILLIAM JAS. PENFOLD,
Director.

The Baker Institute,
Melbourne,
May 16, 1933.

CHRONIC HYPOGLYCÆMIA.

SIR: IN THE MEDICAL JOURNAL OF AUSTRALIA of Saturday, February 18, 1933, there appears an article on "Chronic Hypoglycæmia" which merits certain comment. In an attempt to establish this clinical entity as a common factor in a heterogeneous group of cases, the authors would appear to have made assumptions which seem hardly justified by the facts which they present.

The blood sugar of the normal human being in the fasting state is known to vary within certain limits. Further variations in so-called normal values occur because of variations in the several methods which may be employed to estimate the blood sugar and because of the different values obtained from blood collected from different situations. In the series of cases reported, the blood sugar, in many instances, is on the low side of normal, but no mention is made of the biochemical method by which the values were obtained.

If we are to assume that "any reading below 0·09% in the presence of typical symptoms must be regarded with suspicion" is to be diagnostic of hypoglycæmia, then the cases described may possibly be placed in this category. However, care must be taken with sweeping generalizations, lest they become misleading and, in my opinion, this assumption is not justified from the evidence which appears in this paper.

Certain details merit further criticism. It is stated that a diet deficient in carbohydrate and containing large quantities of protein and fat is a factor in producing hypoglycæmia. No evidence supports this statement, which ignores the experimental work of Southwood and others who have shown that diets of this type have, if anything, the reverse effect.

Case I in the series is stated to have suffered from hypoglycæmia because of the excessive loss of lactose during lactation. I know of no reference in medical literature which suggests that lactose has any influence on the blood sugar when estimated by any of the usual methods. The account of Case III contains the absolutely unjustified statement that the patient suffered from chronic hypoglycæmia for a period of many years. The only evidence to support this contention is one solitary estimation of the fasting blood sugar.

With regard to nomenclature. The classification which is suggested would seem to be entirely symptomatic. It is possible that as further cases are thought to be hypoglycæmic this classification will have to be expanded until it reaches gigantic proportions. Already twelve groups or sub-groups are required to classify twenty-five cases. The basis of any classification should surely rest upon causation and no scientific attempt has been made to do this. The occurrence of a low blood sugar reading two hours after the ordinary glucose tolerance test is not unusual. To assume, because of this fact, that certain patients are subject to hypoglycæmia is absurd. If this were true, what justification exists for the treatment of these cases with doses of glucose at infrequent intervals and why did they improve? Throughout the paper not one estimation of blood sugar is given at the period in which these patients were considered to be in a hypoglycæmic attack. The only facts considered are those obtained from single estimations of the fasting blood sugar or from glucose tolerance tests.

The authors go further. In two cases, Numbers 21 and 22, in which the blood sugar curves did not apparently fall low enough, the term "simple relative hypoglycæmia" is applied in order that such patients may be classified as hypoglycæmic.

The mere fact that glucose by mouth improved certain symptoms in this group of patients cannot justify the assumption of "chronic hypoglycæmia" when such inadequate and incomplete biochemical investigations have been performed.

In opening the paper the statement is made that "the growth of knowledge takes place either as an imperceptible rising tide or as oncoming waves, which, by their impact, rapidly erase old landmarks". It is sincerely to be hoped that this wave of enthusiasm will not obliterate the patiently erected scientific landmarks of our advancing knowledge of the subject of carbohydrate metabolism.

Yours, etc.,

EWEN DOWNIE.

32, Collins Street,
Melbourne,
May 22, 1933.

Post-Graduate Work.

A POST-GRADUATE COURSE AT LISMORE.

THE New South Wales Permanent Post-Graduate Committee announces that a week-end course will be held at Lismore, New South Wales, on Saturday, August 5, and Sunday, August 6, 1933, following the general meeting of the North-Eastern Medical Association. Medical practitioners desirous of obtaining further information should communicate with Dr. J. R. Ryan, Lismore.

Corrigendum.

IN the abstracts on ophthalmology published in the issue of May 27, 1933, there are two errors. In the abstract on detachment of the retina the strength of acetic acid used as a neutralizing agent is given as 5%; it should be 0·5%. In the second column, four lines from the top of the column, the word choroid should be cornea.

NOTICE.

Preparation of Snake Venom for the Treatment of Epilepsy.

IN an article on the investigation of the toxicity and sterility of a commercial preparation containing modified snake venom, published in our issue of May 13, at page 581, reference was made to three sporing organisms having been isolated from a commercial preparation of modified viperine venom. We understand that a similar preparation for the treatment of epilepsy is now being prepared

by the Commonwealth Serum Laboratories, and we have been asked to state that the samples referred to in the article were not prepared by the Commonwealth Serum Laboratories.

AUSTRALIAN ARMY MEDICAL CORPS' DINNER.

THE officers' mess of the Australian Army Medical Corps of the Second Military District will hold a mess dinner at 7 o'clock p.m. on Saturday, June 24, 1933, at the Imperial Service Club, O'Connell Street, Sydney. All members of the Corps are urged to attend. The price of tickets is six shillings and dress will be plain clothes.

Books Received.

MINOR MALADIES AND THEIR TREATMENT, by L. Williams, M.D.; Sixth Edition; 1933. London: Baillière, Tindall and Cox; Australia: Angus and Robertson, Limited. Crown 8vo., pp. 433. Price: 10s. 6d. net.

THE EARLY DIAGNOSIS OF THE ACUTE ABDOMEN, by Z. Cope, B.A., M.D., M.S., F.R.C.S.; Sixth Edition; 1932. London: Humphrey Milford (Oxford University Press); Australia: Angus and Robertson, Limited. Demy 8vo., pp. 262, with illustrations.

THE DIABETIC LIFE, ITS CONTROL BY DIET AND INSULIN: A CONCISE PRACTICAL MANUAL FOR PRACTITIONERS AND PATIENTS, by R. D. Lawrence, M.A., M.D., F.R.C.P.; Seventh Edition; 1933. London: J. and A. Churchill; Australia: Angus and Robertson. Demy 8vo., pp. 226.

A HANDBOOK OF EXPERIMENTAL PATHOLOGY, by G. Wagoner, M.D., and R. P. Custer, M.D.; 1933. London: Baillière, Tindall and Cox. Royal 8vo., pp. 176, with illustrations. Price: 23s. net.

DISEASES OF THE SPINAL CORD, by W. B. Cadwalader, M.D., with introduction by W. G. Spiller, M.D.; 1932. London: Baillière, Tindall and Cox. Royal 8vo., pp. 222, with illustrations. Price: 29s. net.

THE DIFFERENTIAL DIAGNOSIS OF ENDOCRINE DISORDERS, by A. W. Rowe; 1933. London: Baillière, Tindall and Cox. Royal 8vo., pp. 230. Price: 23s. net.

ORAL SPIROCHETES AND RELATED ORGANISMS IN FUSO-SPIROCHETAL DISEASE, by D. T. Smith, A.B., M.D.; 1932. London: Baillière, Tindall and Cox. Royal 8vo., pp. 255, with illustrations. Price: 26s. net.

MODERN ASPECTS OF GASTRO-ENTEROLOGY, by M. A. Arafa, M.R.C.P., with foreword by A. F. Hurst, M.D., F.R.C.P.; 1933. London: Baillière, Tindall and Cox. Royal 8vo., pp. 392, with 56 plates. Price: 27s. 6d. net.

Diary for the Month.

JUNE 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 20.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 21.—Western Australian Branch, B.M.A.: Branch.
JUNE 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 23.—Queensland Branch, B.M.A.: Council.
JUNE 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 28.—Victorian Branch, B.M.A.: Council.
JUNE 29.—South Australian Branch, B.M.A.: Branch.
JUNE 29.—New South Wales Branch, B.M.A.: Branch.
JULY 3.—New South Wales Branch, B.M.A.: Organisation and Science Committee.
JULY 4.—New South Wales Branch, B.M.A.: Council, quarterly.
JULY 5.—Western Australian Branch, B.M.A.: Council.
JULY 5.—Victorian Branch, B.M.A.: Branch.
JULY 6.—South Australian Branch, B.M.A.: Council.
JULY 7.—Queensland Branch, B.M.A.: Branch.
JULY 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers (male).
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephone: MW 2651-2.)

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